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IT LOOKS LIKE A BIG YEAR

The year 1934 promises to be a memorable one as far as the veterinary profession in the United States is concerned. The big event, of course, will be the Twelfth International Veterinary Congress, in New York, in August. This event will be held in conjunction with the 71st annual convention of the American Veterinary Medical Association, the dates for which have been fixed officially, by the Executive Board, as August 14-15-16, 1934. The JOURNAL will carry announcements concerning the big New York meeting, from month to month as the plans are developed.

Another event that will occur during 1934 is the fiftieth anniversary of the establishment of the United States Bureau of Animal Industry by Act of Congress. This occurred on May 24, 1884, about a year after the creation of the Veterinary Division in the Department of Agriculture by Dr. D. E. Salmon. It is very fitting that the International Veterinary Congress should meet in the United States, for the first time, during the year that our Bureau of Animal Industry marks its fiftieth birthday. Right here it would be in order to announce that Honorable Henry A. Wallace, Secretary of Agriculture, has accepted the invitation to serve as Vice-Patron to the Twelfth International Veterinary Congress.

Two of our veterinary colleges will have opportunities to celebrate, during 1934, the fiftieth anniversaries of their founding.

Preliminary announcements have already appeared to the effect that the University of Pennsylvania School of Veterinary Medicine will celebrate the fiftieth anniversary of the opening of the School, in October. As a part of the occasion it is planned to publish a book containing a history of the School and a list of the alumni with their achievements. If our records are correct, the College of Veterinary Medicine of Ohio State University opened its doors in 1884. However, no information has reached us to the effect that the fiftieth anniversary would be marked in any particular way.

At least one state association will hold its fiftieth anniversary meeting this year. The Veterinary Medical Association of New Jersey, as announced in the December issue of the JOURNAL, will meet in Newark on January 4 and 5, to celebrate its golden anniversary in proper style.

So, it looks like a big year for the profession. Let us all make the most of it.

PRESIDENT FITCH OUT FOR A RECORD

Judging from the number of invitations already accepted by President Fitch, he is out for a record, as far as A. V. M. A. presidents and their attendance at veterinary meetings are concerned.

In October, Dr. Fitch attended meetings of the Inter-State Veterinary Medical Association, in Sioux City, and the Central Nebraska Veterinary Association, at Kearney. November 23-24 found him in Atlanta for the annual meeting of the Southern States Veterinary Medical Association.

The first week in December, President Fitch was in Chicago for the meetings of the A. V. M. A. Executive Board, the U. S. Live Stock Sanitary Association and the State and Provincial Research Workers in Animal Diseases. The following week he attended the meetings of the Nebraska State Veterinary Medical Association, in Lincoln, and the South Dakota Veterinary Medical Association, in Sioux Falls.

For January, Dr. Fitch is scheduled to attend the 50th Anniversary meeting of the Veterinary Medical Association of New Jersey, at Newark, on the 4th and 5th. Thence he will go to Philadelphia, for the Conference of Veterinarians of the University of Pennsylvania, on the 9th and 10th. The next two days (January 11-12) will be spent at Ithaca, N. Y., attending the Cornell Conference. The following week (January 16) he

will be in Indianapolis, for the meeting of the Indiana Veterinary Medical Association, and in Manhattan for the Kansas Veterinary Medical Association meeting (January 17-18). The fourth week in January Dr. Fitch will be busy with the meeting (January 25-26) in his own state. He hopes to be able to spend a day at the Iowa meeting in Des Moines (January 23-25), and a day at the Wisconsin meeting, in Madison (January 22-24). In all probability he will go to Columbia, for the University of Missouri Short Course, the last week of the month.

PRAISE FOR TWO EXTENSION DIRECTORS

Two instances of a very apparent desire to coöperate with veterinarians, upon the part of state directors of extension work, have recently been brought to our attention. The first of these came from Michigan, a state where there has been comparatively little trouble during recent years. The satisfactory situation in Michigan undoubtedly was brought about very largely through the coöperative efforts of the members of the extension service of Michigan State College and the Michigan State Veterinary Medical Association. As a result of the united efforts of these two organizations, a working agreement was drawn up, several years ago, and this soon brought about a better understanding upon the part of both extension workers and veterinarians.

However, in matters of this kind, affecting a considerable number of individuals, it is always an easy matter to forget something that has been agreed upon, unless there are more or less constant reminders of the existence of an agreement. This appears to have been the case in Michigan. As a result, Mr. R. J. Baldwin, director of extension work at Michigan State College, under date of October 28, 1933, sent the following letter to all county agricultural agents in Michigan:

> On several occasions during the past few months, the question of relationship between the local veterinarian and the county agricultural agent has been brought up for discussion. This matter was referred to a committee, chosen from the college staff in 1931, and that committee recommended certain policies as guides to extension workers and their report was sent to the county agricultural agents at that time. In order that you may refresh your memory regarding this report, I am sending a copy of it to you at this time.
>
> I wish to emphasize again the necessity for working in close harmony with the veterinary profession, and urge that you coöperate with the veterinarians in all projects relating to the health of farm animals and invite them to take part in educational demonstrations, such as sheep drenching and other projects of a similar nature.

The letter speaks for itself. This incident illustrates the desirability of keeping such things fresh wherever a large number of individuals are parties to an agreement. In the absence of anything in the shape of a reminder, it is so easy to forget. We make the suggestion to Director Baldwin that it might not be a bad idea to send out such a letter annually, merely as a reminder to the county agents in Michigan that there is such a thing as a working agreement between extension workers and practicing veterinarians. The officers of the Michigan State Veterinary Medical Association might do the same thing, in a spirit of fair play. Thanks to you, Director Baldwin.

The other incident happened in Missouri. One of the unethical serum companies mailed a letter to the county agents in Missouri, and we are reproducing the first two paragraphs of the letter:

> Today farmers universally vaccinate and treat their live stock themselves. This eliminates the cost of veterinary service.
>
> County extension agents are our dealers. We allow you a 10 per cent discount on hog cholera virus and a 20 per cent discount on anti-hog cholera serum and other products.

Mr. R. R. Thomasson, assistant director in charge of extension work at the University of Missouri, has shown himself to be a pretty good friend of the veterinarian on numerous occasions in the past. When the letter sent to county agents, by the unethical serum company, was shown to Mr. Thomasson, he promptly addressed a letter to the president of the company and proceeded to explain that county agents can not act as agents for the sale of serum or any such product. We quote the first paragraph of Mr. Thomasson's letter:

> Some of our county agents have sent me copies of your recent letter to them regarding the distribution of hog cholera serum and virus. I am sure that this letter was written without a complete understanding of the conditions and regulations under which county agents operate. County extension agents in Missouri are not permitted to act as agents for any commercial product or concern. Occasionally in introducing a new product they place orders for a few farmers until a source of supply can be established. In doing so, however, they do not charge a commission. It would be unethical and a violation of the policy of this institution for any county agent to charge a commission or make a profit on any commodity that he might order for his people.

The unfortunate part of this particular case is to be found in the fact that the president of the serum company, above referred to, was encouraging county agents to engage in something that has been strictly forbidden, not only by the University of Missouri, but by the Secretary of Agriculture of the United States. We heartily commend your action, Mr. Thomasson. It is definitely in the right direction.

TWO MORE STATES AFFILIATE

At the annual meeting of the Arizona State Veterinary Medical Association, held in Phoenix, on December 15, the organization voted to affiliate with the A. V. M. A. On the same day, the South Dakota Veterinary Medical Association, meeting in Sioux Falls, took the same action, bringing the number of affiliated state associations up to 37.

In the December issue of the JOURNAL, we inadvertently included Vermont in the list of states that had not affiliated. Dr. G. N. Welch, secretary of the Vermont Veterinary Medical Association, draws our attention to the fact that Vermont voted to affiliate at the 1932 summer meeting. West Virginia should have been included in the list instead of Vermont. We regret the error.

The action of the Arizona and South Dakota associations reduces to ten the number of state organizations that have not voted to affiliate. The correct list now is as follows: Alabama, Arkansas, Indiana, Iowa, Mississippi, New Hampshire, New Mexico, Rhode Island, West Virginia and Wyoming.

HONOR ROLL

At the Chicago convention, the Association adopted an amendment to the By-laws creating an Honor Roll, to consist of those who have been active members of the Association for a period of fifty years. These members continue to share all the rights and privileges of active membership, but without the further payment of dues. The following comprise the first Honor Roll:

Dr. C. W. Crowley (1876), Saint Louis, Mo.

Dr. Benjamin McInnes (1876), Charleston, S. C.

Dr. L. H. Howard (1882), Brookline, Mass.

Membership cards for 1934 have been issued to these three members, who have paid dues each year since their admission to membership, shown in parentheses in each case.

EXECUTIVE BOARD ELECTION

The special election now in progress in Executive District 5 will come to a close on January 13. Unusual interest is being shown in this election. District 5 consists of only two states—Iowa and Minnesota—and each state is very desirous of electing a representative to the Board. The successful candidate will

fill out the unexpired term of Dr. C. P. Fitch. The indications are that a higher percentage of the available votes will be cast in this election than in any Executive Board election held in recent years.

OHIO VETERINARY CONFERENCE

Veterinarians of Ohio and adjoining states will welcome the announcement, recently made by Dean Brumley, to the effect that the annual Veterinary Conference at the Ohio State University, College of Veterinary Medicine, would be resumed in 1934. The 1933 Conference was not held, on account of the economic depression. The dates have been fixed for March 21-22-23. A more extended announcement will be made in the JOURNAL at a later date.

APPLICATIONS FOR MEMBERSHIP

An even hundred applications for membership were filed during 1933. Losses through death and resignation almost reached the hundred mark. About 250 members were dropped from the roll for the non-payment of dues, less than 6 per cent of the membership as shown by the 1932 report. How many national organizations can show as good a record for the same period?

There is one good feature about the new members of the past year. In practically every case they joined without the urge of a high-pressure membership campaign. In many cases an application was the culmination of correspondence with the Secretary on one subject or another. The point we would like to make here is that these new members joined because they wanted to, and not because some other member wanted them to join. These new members felt that the Association had something for them. They joined to get the privileges of membership and for the enjoyment that they would get from being a part of a large, worthwhile organization. This is as it should be.

At a recent conference of organization officials, it was practically the unanimous opinion that new members obtained in so-called membership "drives" do not "stick," in a large percentage of cases. A study of A. V. M. A. records bears out this contention to a certain degree. A steady, consistent growth is much to be desired over a sudden, spectacular increase in membership. During times such as we have had during the past four years, even "holding one's own" is an accomplishment.

Every six months we remind our members of the modus operandi for joining the A. V. M. A. We find that this saves us much correspondence. The rules are simple. Here they are:

Applications for membership shall be made upon blanks furnished by the Association in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Applications must be accompanied by a membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Applications must be filed with the Secretary and examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereafter as possible, said application with name and address of the applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary and membership card issued. If any objections be filed against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

BARTA, FRED — De Witt, Nebr.
D. V. M., Kansas City Veterinary College, 1917
Vouchers: E. C. Jones and Frank Breed.

GEISLER, R. E. — 50 Cooper Sq., New York, N. Y.
D. V. M., Iowa State College, 1932
Vouchers: D. H. Udall and H. J. Milks.

HUDSON, H. K. — 521 Convery Place, Perth Amboy, N. J.
D. V. M., Kansas State College, 1933
Vouchers: M. L. Morris and E. R. Frank

STOUDT, M. D. — 4818 Frankford Ave., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1933
Vouchers: M. A. Emmerson and C. J. Marshall.

WEST, J. R. — Animal Diseases Research Institute, Hull, Que., Can.
B. V. Sc., Ontario Veterinary College, 1930
Vouchers: Chas. C. Mitchell and C. W. McIntosh.

Application Pending

SECOND LISTING

(See December, 1933, JOURNAL)

McGinnis, C. L., Far Hills, N. J.

The amount which should accompany an application filed this month is $10.00, which covers membership fee and dues to January 1, 1935, including subscription to the JOURNAL.

STATE BOARD EXAMINATION

Iowa Veterinary Medical Examining Board. State House, Des Moines, Iowa. January 22-23, 1934. Candidates for examination must be in the office of the Division of Animal Industry not later than 9:00 a. m., January 22. Dr. Peter Malcolm, Secretary, State House, Des Moines, Iowa.

COMING VETERINARY MEETINGS

California State Veterinary Medical Association and University of California Veterinary Conference. University Farm, Davis, Calif. January 2-5, 1934. Dr. Geo. M. Simmons, Secretary, 1386 Golden Gate Ave., San Francisco, Calif.

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. and 103rd St., New York, N. Y. January 3, 1934. Dr. John E. Crawford, Secretary, 708 Beach 19th St., Far Rockaway, Long Island, N. Y.

Western Michigan Veterinary Medical Association. Grand Rapids, Mich. January 4, 1934. Dr. C. H. Haasjes, Secretary, 728 S. State St., Shelby, Mich.

New Jersey, Veterinary Medical Association of. Hotel Douglas, Newark, N. J. January 4-5, 1934. Dr. John G. Hardenbergh, Secretary, c/o Walker-Gordon Lab. Co., Plainsboro, N. J.

Interstate Veterinary Medical Association. Elks Building, Omaha, Neb. January 8, 1934. Dr. G. L. Taylor, Secretary, Plattsmouth, Neb.

Oklahoma Veterinary Medical Association. Biltmore Hotel, Oklahoma City, Okla. January 8-9, 1934. Dr. C. H. Fauks, Secretary, 1719 S. W. 15th St., Oklahoma City, Okla.

Intermountain Livestock Sanitary Association. Ogden, Utah. January 8-10, 1934. Dr. D. E. Madsen, Secretary, Utah Experiment Station, Logan, Utah.

Chicago Veterinary Medical Society. Hotel La Salle, Chicago, Ill. January 9, 1934. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

Northwestern Missouri Veterinary Medical Association. Maryville, Mo. January 9, 1934. Dr. R. L. Cundall, Secretary, Fairfax, Mo.

San Diego County Veterinary Medical Association. San Diego, Calif. January 9, 1934. Dr. L. K. Knighton, Secretary, 3438 Mountain View, San Diego, Calif.

Pennsylvania, Conference for Veterinarians at University of. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 9-10, 1934. Dr. G. A. Dick, Dean, 39th St. and Woodland Ave., Philadelphia, Pa.

Maine Veterinary Medical Association. State House, Augusta, Me. January 10, 1934. Dr. L. E. Maddocks, Secretary, R. F. D. 2, Augusta, Me.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. January 10, 1934. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.

Willamette Valley Veterinary Medical Association. Woodburn, Ore. January 10, 1934. Dr. E. W. Coon, Secretary, Forest Grove, Ore.

Tennessee Veterinary Medical Association. Patton Hotel, Chattanooga, Tenn. January 10-11, 1934. Dr. A. C. Topmiller, Secretary, Box 238, Murfreesboro, Tenn.

Tulsa County Veterinary Association. Tulsa, Okla. January 11, 1934. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

Cornell University, Annual Conference for Veterinarians at. New York State Veterinary College, Ithaca, N. Y. January 11-12, 1934. Dr. W. A. Hagan, Dean, Cornell University, Ithaca, N. Y.

Texas, State Veterinary Medical Association of. Kyle Hotel, Temple, Tex. January 15-16, 1934. Dr. D. Pearce, Secretary, Box 335, Leonard, Tex.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. January 16, 1934. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Indiana Veterinary Medical Association. Severin Hotel, Indianapolis, Ind. January 16-18, 1934. Dr. W. B. Craig, Secretary, 1420 N. Alabama St., Indianapolis, Ind.

Kansas Veterinary Medical Association and Kansas State College Conference for Veterinarians. Kansas State College, Manhattan, Kan. January 17-18, 1934. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kan.

Colorado Veterinary Medical Association. Albany Hotel, Denver, Colo. January 18, 1934. Dr. Floyd Cross, Secretary, 711 Mathews Ave., Fort Collins, Colo.

Mississippi State Veterinary Medical Association. Greenwood, Miss. January 18-19, 1934. Dr. H. H. Collins, Secretary, Laurel, Miss.

Vermont Veterinary Medical Association. Montpelier Tavern, Montpelier, Vt. January 19, 1934. Dr. G. N. Welch, Secretary, 43 Union St., Northfield, Vt.

Washington State Veterinary Medical Association, Western Branch of the. Tacoma, Wash. January 19, 1934. Dr. Clifford Ackley, Secretary, Winlock, Wash.

South Carolina Association of Veterinarians. Jefferson Hotel, Columbia, S. C. January 22, 1934. Dr. G. J. Lawhon, Secretary, Hartsville, S. C.

Wisconsin Veterinary Medical Association. Park Hotel, Madison, Wis. January 22-24, 1934. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Michigan State College Short Course for Veterinarians. Michigan State College, East Lansing, Mich. January 22-26, 1934. Dr. Ward Giltner, Dean, Michigan State College, East Lansing, Mich.

Iowa Veterinary Medical Association. Hotel Savery, Des Moines, Iowa. January 23-25, 1934. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

Massachusetts Veterinary Association. Hotel Westminster, Boston, Mass. January 24, 1934 (annual). Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Ontario Veterinary Association. Royal York Hotel, Toronto, Ont. January 24-25, 1934. Dr. H. M. LeGard, Secretary, 335 N. Main St., Weston, Ont.

Minnesota State Veterinary Medical Society. Saint Francis Hotel, Saint Paul, Minn. January 25-26, 1934. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Ohio State Veterinary Medical Association. Neil House, Columbus, Ohio. January 25-26, 1934. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Nevada State Veterinary Association. University of Nevada, Reno, Nev. January 30, 1934. Dr. Warren B. Earl, Secretary, Box 1027, Reno, Nev.

Missouri Veterinary Medical Association and Special Course for Graduate Veterinarians. University of Missouri, Columbia, Mo. January 30-31, February 1, 1934. Dr. Ashe Lockhart, Secretary, 800 Woodswether Rd., Kansas City, Mo.

Alabama Veterinary Medical Association and Short Course for Graduate Veterinarians. College of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala. February 5-10, 1934. Dr. C. A. Cary, Dean, Alabama Polytechnic Institute, Auburn, Ala.

Connecticut Veterinary Medical Association. Hotel Garde, Hartford, Conn. February 7, 1934. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

Hudson Valley Veterinary Medical Society. Albany, N. Y. February 14, 1934. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

THE PRESENT STATUS OF ANTHELMINTIC MEDICATION FOR GASTROINTESTINAL PARASITES OF THE HORSE*

By WILLARD H. WRIGHT, *Washington, D. C.*
Zoölogical Division, Bureau of Animal Industry
United States Department of Agriculture

INTRODUCTION

During the past few years, veterinarians and horse-owners have shown an awakened interest in the control of internal parasites of equines. The very successful campaigns inaugurated in Iowa, by the Iowa Veterinary Medical Association, and in Illinois, by Graham and his associates, both campaigns involving the coöperation of state and federal extension services, horse-owners and practicing veterinarians, have shown what can be accomplished by coöperative effort and will no doubt point the way to similar campaigns in other states. The growing realization of the ravages of horse parasites began to be felt at a time when the horse was perhaps at its lowest level of economic importance. Now that the tide apparently has turned toward a wider utilization of the horse as a source of farm power, this increased realization of the damage and economic loss due to parasites should be made the basis for increased effort to develop and apply measures for the control of horse parasites. The control of these parasites involves many factors other than anthelmintic medication, but it is the purpose of this paper to discuss only the factor of treatment. It is this factor with which the veterinary practitioner is mostly concerned.

Prior to the work of Hall and his associates, anthelmintic medication for equine parasites was largely on an empirical basis. Outside of the carbon disulfid treatment for the removal of bots, we had no well established knowledge of any dependably effective anthelmintic. In the materia medica of those days, one finds recommendations for the use of such drugs as iron sulfate, arsenic, copper sulfate and tartar emetic, mixed with the feed. These drugs have their proper place in certain kinds of therapy, but, as administered to horses to remove worms, they are almost entirely useless as far as any parasiticidal action is concerned. Experiments carried out over the past fifteen years have furnished us with dependably effective anthelmintics for nearly all of the gastrointestinal parasites of the horse.

*Presented at the sixty-ninth annual meeting of the American Veterinary Medical Association, Atlanta, Ga., August 23-26, 1932.

The necessity for anthelmintic medication is indicated either by clinical observation or by microscopic examination of the feces for worm eggs. Unthriftiness that is not obviously a result of poor feeding or poor breeding should lead the clinician to suspect parasitism as a cause, and a tentative clinical diagnosis of parasitism should be checked by a laboratory examination for confirmation. Microscopic examination will usually indicate the approximate degree of infestation with most nematode parasites, and, to a lesser extent, with tapeworms. It is obviously of no value for the diagnosis of bot infestations. The species of parasites present will determine the anthelmintic to be used, with the reservations dictated by the physical condition of the animal and the presence of contraindications for any particular drug.

Judging from the number of inquiries on the subject, there is a considerable demand for an anthelmintic mixture which will be effective against both bots and most nematode parasites. That such an anthelmintic mixture would be of decided advantage is fully recognized, but up to the present time we do not know of any one drug or combination of drugs which is effective both for the removal of bots and ascarids on the one hand and strongyles and cylicostomes on the other.

Method of Administration of Anthelmintics

The administration of drugs by means of the stomach-tube is the preferred method. In the case of highly volatile drugs, such as carbon disulfid, the use of the stomach-tube obviates the danger of breakage of capsules in the mouth and inhalation of the drug. Fatalities from this cause are not frequent, it is true, but such accidents, even without fatalities, make a bad impression on the owner and reflect upon the professional ability and skill of the veterinarian. In horse-parasite-control campaigns, where large numbers of horses are to be treated daily, the procedure of passing the stomach-tube usually would be too time-consuming, but in all ordinary anthelmintic medication, the use of the stomach-tube would be the method of choice.

Prior to the administration of anthelmintics, animals should be fasted for the periods indicated under the various treatments described in this paper. For parasites of the stomach and small intestine, fasting for 18 to 24 hours is usually sufficient. However, in treatment for the removal of strongyles, cylicostomes and oxyurids from the large intestine, a longer period of fasting is necessary to reduce the bulk of contents in the stomach and small intestine, and to permit the anthelmintic to reach the cecum and colon expeditiously and in the greatest possible concentra-

tion. For parasites in the large intestine, experience indicates that the animal should be fasted for 36 hours. Water may be allowed during the period of fasting, but both food and water should be withheld for four to five hours after the administration of the anthelmintic.

Time of Treatment

In order to keep parasitic infestation at a minimum and avoid losses from this source, it is advisable to adopt a program of regular treatment. The usual procedure is to administer treatments twice a year. In the northern part of the United States, animals may be treated for worm parasites in the late spring or early summer and in the fall, and for bots in late November or December and again in February, if necessary, to remove those which have developed since the first treatment. In the South, owing to the shorter and milder winter and the more abundant warmth and moisture, it may be necessary to treat oftener. However, judgment, based on experience, is a better basis for procedure than any general rule. It is not advisable to administer any treatment oftener than once in four to six weeks. Where only one treatment is to be administered for bots, it should be given early in February in the northern United States, and late in February in the southern United States. It is known that the larvae of at least one species of bot found in the United States, namely, *Gasterophilus intestinalis*, do not migrate directly to the stomach but spend some time in the mucous membrane of the cheek and tongue. It is likely, therefore, that a single treatment administered in December will fail to destroy all bots present in the animal.

Contraindications for Treatment

Contraindications for the use of anthelmintics are general and specific. General contraindications are those which involve the physical condition of the animal and the presence of diseases not parasitic in origin. Very young or very old animals or those in a weakened condition are poor risks for anthelmintic medication, and, as a rule, anthelmintics should not be administered to animals suffering from febrile conditions. The treatment of animals suffering from severe parasitic infestations resulting in a state of extreme debility presents a problem for the judgment of the veterinarian. The use of an anthelmintic in such animals may be sufficient to turn the tide toward an unfavorable conclusion or it may result in striking improvement. Whether such animals should be treated with the standard dose of a suitable anthelmintic, whether they should be given divided doses over a period

of time, or whether an effort should be made to build up the physical condition of the animal before treatment is undertaken are questions which judgment, based on experience, alone will answer.

Specific contraindications exist for nearly all drugs used in the treatment of equine parasitisms and a knowledge of these contraindications on the part of the practitioner frequently will obviate unfavorable reactions to treatment. These contraindications will be taken up in detail.

Carbon disulfid: This drug exerts a marked irritating action on the mucosa of the stomach and upper small intestine. Animals killed shortly after treatment with carbon disulfid show lesions of a degree ranging from a marked reddening to actual vesication of the mucosa. Carbon disulfid, therefore, should not be given to animals suffering from gastroenteritis. Carbon disulfid occasionally produces colicky pains of a spasmodic type. Administered when gastric or intestinal colic is present, the drug, no doubt, would greatly aggravate the condition. The advisability of administering this drug to pregnant mares is debatable. Many practitioners in area work in horse parasite control have not hesitated to use the drug on mares due to foal the following spring. However, on account of the gastrointestinal irritation caused by the drug and the occasional symptoms of colic manifested following its administration, treatment should be withheld from mares in advanced pregnancy. Fats and oils should not be given with carbon disulfid, as such substances promote the absorption of the drug and lead to marked systemic reactions. In case of breakage in the mouth of capsules containing carbon disulfid, the mouth should be washed out with water immediately, as the drug is only slightly soluble in water. The head of the animal should be held down to prevent aspiration of the drug into the trachea. Plenty of fresh air should be provided, and respiratory and general stimulants should be administered freely, if indicated.

Carbon tetrachlorid: Carbon tetrachlorid in therapeutic doses usually produces a central necrosis in the cells of the liver acini. Consequently the drug is contraindicated in the presence of icterus, hepatitis or cirrhosis of the liver. Cases have been reported in children and in puppies where the administration of carbon tetrachlorid in the presence of a heavy ascarid infestation has produced clumping of the ascarids in the intestinal tract with subsequent enteritis, toxemia and death. While such a phenomenon has not been reported following the administration of carbon tetrachlorid to colts, the possibility of its occurrence

should be kept in mind. Therefore, in treating colts heavily infested with ascarids it is advisable to accompany the carbon tetrachlorid with a saline purgative.

The retention of bile pigments resulting from acute hepatic injury following the administration of carbon tetrachlorid appears to have a decided influence upon calcium metabolism. Minot[1] observed that small doses of this drug caused severe and usually fatal intoxication in dogs on a diet high in protein and low in calcium, while Lamson and his associates[2] found that carbon tetrachlorid, even in very large doses, produced no acute intoxication in dogs kept on a well-balanced mixed diet. Minot and Cutler[3] found in cases of intoxication that the bilirubinemia increased progressively during the course of the intoxication, and that there was a corresponding increase in guanidine concentration in the blood. The increase in guanidine paralleled the severity of the symptoms, and the administration of guanidine produced an intoxication very similar to that caused by carbon tetrachlorid. It is believed that the effect of calcium may lie in its antagonistic effect upon the retained guanidine, since the addition of calcium salts to the diet prior to the administration of carbon tetrachlorid or guanidine prevented the development of toxic manifestations. Furthermore, calcium therapy during the course of these manifestations resulted in prompt cessation of symptoms.

After treating a considerable number of animals in the clinic of the Berlin Veterinary College, Neumann-Kleinpaul and Pelckmann,[4] mention the following symptoms produced by the administration of carbon tetrachlorid: apathy, anorexia, increased thirst, colicky symptoms, tremor, bloating, evidences of pain, fluctuating temperature, variation in the pulse and respiration rates, cough, diarrhea, icterus, and the appearance of albumin in the urine. They found that the degree of toxicity is not always in proportion to the size of the dose of carbon tetrachlorid. Furthermore, they demonstrated that the blood-calcium level, computed from the daily average, sank from 4 to 14.56 per cent below its original level after the administration of carbon tetrachlorid in doses of 0.15 to 0.33 gm per kilogram of body weight, indicating that the relation of calcium to the toxicity of carbon tetrachlorid in horses is substantially the same as in dogs, as demonstrated by Minot. Grassnickel[5] found that the bilirubin content of the blood was increased 1.2 to 4 times in every case after carbon tetrachlorid administration. We have obtained practically parallel results in a study of the serum calcium, phosphorus and

bilirubin level of the blood following the administration of carbon tetrachlorid to horses.

In substance, the above-mentioned facts indicate that the horse is no less susceptible to carbon tetrachlorid intoxication than are other animals and that, before the administration of carbon tetrachlorid, the diet of the horse should be regulated in such a way as to provide a high calcium intake in order to offset lowering of the blood-calcium level and to prevent intoxication. Animals suffering from hepatic disease or from calcium deficiencies, such as rickets or osteomalacia, are poor risks for carbon tetrachlorid therapy.

The following therapy is indicated in the treatment of carbon tetrachlorid intoxication in the horse: In severe intoxications, where immediate relief is necessary to prevent death, the intravenous administration of 75 to 150 cc of a 10 per cent solution of calcium gluconate is indicated. The injection should be made very slowly. There is a marked increase in the serum calcium of the blood within five to ten minutes, the pre-injection level being regained usually within several hours. In subacute intoxications, where more delayed absorption of calcium is indicated, calcium gluconate should be given intramuscularly in a dose of 100 to 150 cc of the 10 per cent solution. Following intramuscular injection, the blood calcium may recede to its previous level in six to eight hours and treatment should be repeated, if indicated. Parathyroid hormone, administered intramuscularly in a dose of 400 to 500 units for a 1,000-pound animal, will accomplish the same effect. Other less satisfactory methods of therapy are the oral administration of calcium chlorid or lactate in a dose of one-half ounce (15 grams) three times a day, or ammonium chlorid in a dose of 2 drams (8 grams) three times a day in conjunction with calcium salts. Calcium chlorid is more irritating than is the lactate and should not be given for over six to eight doses at any one time. Ammonium chlorid is thought to increase hydrogen-ion concentration and promote the ionization of calcium.

Experiments to determine the effect of specific therapy in carbon tetrachlorid intoxication in the horse were carried out by the writer and John Bozicevich, of the Zoölogical Division. Table I presents the results of parathyroid hormone therapy; table II presents the results of calcium gluconate therapy. In each of these experiments it will be seen that there was a marked decrease in the blood-calcium level following the administration of carbon tetrachlorid in a dose of 0.3 cc per kilogram of body weight, with a concomitant decrease in the blood-phosphorus level and a marked hyperbilirubinemia. Following the administration of the

TABLE I—*The effect of parathyroid hormone therapy on the blood-calcium level in carbon tetrachlorid intoxication in the horse. (Horse 1, gray mare, 15 years old, weight 565.9 kilograms)*

DATE (1932)	HOUR	TREATMENT	CALCIUM (MG. PER 100 CC OF SERUM)	INORGANIC PHOSPHORUS (MG. PER 100 CC OF SERUM)	BILIRUBIN (MG. FOR 100 CC OF SERUM)	SYMPTOMS
8–8	9 a.m.		11.0	3.3	0.6	
	3 p.m.		10.7	3.1	0.6	
8–9	9 a.m.		11.5	3.0	0.6	
	9:30 a.m.	0.3 cc per kilogram of carbon tetrachlorid				
	3 p.m.		9.5	3.1	0.65	
8–10	9 a.m.		10.0	2.7	1.15	Anorexia, dizziness, marked intoxication
	3 p.m.		10.5	2.2	1.35	
	3:30 p.m.	500 units Para-thor-mone (Lilly) intramuscularly				
8–11	9 a.m.		11.5	2.3	1.75	
	3 p.m.		12.0	2.5	1.75	Marked improvement, appetite returned, animal eating well
8–12	9 a.m.		12.0	2.7	1.25	Animal showed continued improvement
	3 p.m.		11.0	2.7	1.25	

TABLE II—*The effect of calcium gluconate therapy on the blood-calcium level in carbon tetrachlorid intoxication in the horse. (Horse 2, black gelding, 5 years old, weight 863.63 kilograms.)*

DATE (1932)	HOUR	TREATMENT	CALCIUM (MG. PER 100 CC OF SERUM)	INORGANIC PHOSPHORUS (MG. PER 100 CC OF SERUM)	BILIRUBIN (MG. PER 100 CC OF SERUM)	SYMPTOMS
8–8	9 a.m.		13.0	3.2	0.25	
	3 p.m.		12.0	3.2	0.40	
8–9	9 a.m.		11.0	3.3	0.35	
	9:30 a.m.	0.3 cc per kilogram of carbon tetrachlorid				
	3 p.m.		9.5	3.0	0.50	
8–10	9 a.m.		10.5	3.0	1.25	Anorexia, staggering gait, marked distress
	3 p.m.		10.5	2.5	1.50	
8–11	9 a.m.		10.5	2.7	1.75	
	9:30 a.m.	150 cc of 10 per cent solution of calcium gluconate (Sandoz) intravenously				
	10:30 a.m.		13.0	2.6	1.75	
	3 p.m.		12.5	2.5	1.75	
	4 p.m.	150 cc of 10 per cent solution of calcium gluconate (Sandoz) intramuscularly				
8-12	9 a.m.		12.5	2.8	1.10	Cleaned up all feed, apparently normal
	3 p.m.		12.0	2.7	1.15	

drug, both animals showed well-marked symptoms of carbon tetrachlorid intoxication. The administration of parathyroid hormone or calcium gluconate, as indicated in the tables, led to a prompt rise in the blood-calcium level and a disappearance of the symptoms of intoxication. As would be expected, the specific therapy in each case had no apparent effect on the blood-phosphorus level or the bilirubin level of the blood.

N-butylidene chlorid: This compound, which was found by Wright and his collaborators[6] to be very effective for the removal of strongyles and cylicostomes from the horse, apparently does not produce the central necrosis of the liver which usually follows the administration of carbon tetrachlorid. Theoretically, it should, therefore, be a safer drug than is carbon tetrachlorid. We have given a horse butylidene chlorid in a dose of 1.5 cc per kilogram of body weight without lethal effect. One animal, which received a dose of 3.0 cc per kilogram of body weight, survived the treatment but later succumbed to pneumonia following exposure. On necropsy this animal showed no lesions of gastroenteritis in spite of the large dose of the drug. In the horse, this drug has the tendency to produce constipation and its administration therefore is contraindicated in the presence of chronic constipation. It is possible that other contraindications will develop as the drug is used more extensively in the treatment of horse parasites. Some preliminary work would seem to indicate that butylidene chlorid may have some effect also on the blood-calcium level, but further work is needed to establish this point.

Oil of chenopodium: This drug is a gastrointestinal irritant and exerts a depressant action on the central nervous system and the nerve endings of the intestinal tract. Oil of chenopodium is contraindicated in the presence of constipation, gastroenteritis and febrile conditions. It always should be preceded or followed immediately by an adequate dose of an active purgative, preferably raw linseed oil. Oil of chenopodium should not be given to pregnant mares.

There is some evidence to indicate that highly bred animals of a nervous temperament do not tolerate oil of chenopodium as well as do phlegmatic animals of the draft breeds. Several cases of laminitis have been reported following the use of the standard dose of oil of chenopodium. Most of these cases have occurred in Thoroughbreds, hunters or saddle horses. The exact mechanism whereby the drug produces this condition is not known but it is possible that the drug acts by depressing the vagus and the vaso-motor center. Absorption of the drug in such cases would seem to be due to insufficient purgation. Where adequate purga-

tion does not follow within the usual time, an additional dose of purgative should be given.

Tetrachlorethylene: This is a much safer drug than is carbon tetrachlorid, but unfortunately the compound possesses relatively little efficacy for the removal of any species of horse parasites. Raffensperger has conducted a number of critical tests with tetrachlorethylene, and the results of these tests, not yet published in detail, indicate in a general way that tetrachlorethylene is decidedly inferior to the other treatments outlined in this paper. Tetrachlorethylene is partially effective for the removal of bots and might be indicated where carbon disulfid is contraindicated.

Specific Therapy

Tapeworms: Tapeworms are apparently not very prevalent in horses in this country. Wehr[7] made a survey of 525 horses in the horse-slaughtering establishment at Miles City, Montana. Of these, 75 harbored *Anoplocephala mamillana* and 2 harbored *Anoplocephala magna*. Hall,[8] in 1918, summarized the occurrence of *Anoplocephala* spp. in the United States and stated that *A. magna*, said to be the most rare of the tapeworms of the horse in Europe, is the commonest of the three species in this country. Hall recorded 12 cases of *A. magna* in the United States up to that time.

Little is known about the treatment for tapeworm infestation in the horse, as there is practically no evidence based on critical tests of drugs on horses infested with tapeworms. Turpentine has been recommended in an initial dose of 2 ounces, followed every second day by 1 ounce for five or six doses, the last dose to be followed by 1 quart of raw linseed oil. Areca nut may be given in a dose of 1 to 1.5 ounces for a 1,000-pound animal. Areca nut should be freshly ground. If purgation does not ensue within four or five hours after the administration of areca nut, it is advisable to give purgatives, as absorption of the drug may lead to untoward reactions. Kamala may be used in a dose of 1 ounce for a 1,000-pound animal. Raffensperger gave a 1,100-pound mule a total of 105 grams, or about 3.5 ounces, of kamala over a period of four days without any ill effect. Oleoresin of male fern is another drug which may be used for tapeworms. The dose is 3 to 6 drams for a 1,000-pound animal, immediately preceded or followed by 1 quart of raw linseed oil.

It is advisable to fast animals for 36 hours before the administration of tenicides. These treatments are contraindicated in very young or very old animals, in those suffering from any

febrile condition, or in those suffering from emaciation or debility. None of the above-mentioned tapeworm remedies should be given to animals suffering from gastroenteritis. These drugs should be used with caution in pregnant mares.

Bots (Gasterophilus spp.): At the present time, liquid carbon disulfid is the best treatment for bots. The dose is 6 fluid drams for a 1,000-pound animal or at a dose rate of 1.5 fluid drams for each 250 pounds of body weight. The animals should be fasted for at least 18 hours prior to treatment. Water may be given during this time but both food and water should be withheld for 4 to 5 hours after treatment. As previously stated, fats and oils should be avoided in connection with the administration of carbon disulfid. The so-called "solid" carbon disulfid capsules in which the drug is adsorbed in magnesium carbonate possess a somewhat lower efficacy than does the liquid drug. Furthermore, some of the drug escapes in the course of time and the capsules become brittle with age. Thorp, James and Graham,[9] in a critical test, secured an efficacy of 80 per cent with these capsules and Bozicevich and Underwood[10] obtained respective efficacies of 94.4, 60 and 94.1 per cent.

Carbon tetrachlorid is not particularly effective against horse bots; in Hall's[11] tests it showed an efficacy of only about 25 per cent in doses of 25 to 50 cc. Raffensperger found that tetrachlorethylene, at a dose rate of 0.15 cc per kilogram of body weight, removed from 50 to 75 per cent of the bots. Tetrachlorethylene may be of value in those cases where carbon disulfid is contraindicated because of the poor condition of the animal.

Stomach worms (Habronema spp.): Wright, Bozicevich and Underwood[12] found that carbon disulfid in a dose of 6 fluid drams for a 1,000-pound animal, preceded by gastric lavage with 8 to 10 liters of a two per cent solution of sodium bicarbonate, was very effective for the destruction of *Habronema muscae* and *H. microstoma.* Carbon disulfid without preliminary lavage gave less favorable results. Apparently the alkaline solution serves to remove excess mucus from the stomach wall and permits the drug to reach the parasites more effectively. Furthermore, the solution seems to give some protection against the irritating action of the carbon disulfid. In the above-mentioned experiments, *H. megastoma* in stomach tumors was not affected by the treatment. It is advisable, though not necessary, to siphon off the sodium bicarbonate solution 5 to 10 minutes after its administration.

Ascarids (Ascaris equorum): Carbon disulfid is very effective for the removal of horse ascarids. The drug is used in the same dose as for bots. Oils should not be given with carbon disulfid.

Carbon tetrachlorid also is an effective treatment against *Ascaris equorum*. The animals should be fasted from 24 to 36 hours and carbon tetrachlorid given in a dose of 6 to 12 fluid drams for a 1,000-pound animal. In treating for heavy infestations, it is advisable to follow carbon tetrachlorid with a saline purgative.

Oil of chenopodium, which is very effective against ascarids in most animals, is curiously of very limited value for the removal of *Ascaris equorum*. Butylidene chlorid is probably effective against horse ascarids.

Strongyles and cylicostomes (Strongylus spp., Trichonema spp. and related genera): It is fortunate that we have available several very effective drugs for the treatment of infestations of the horse with large and small strongyles. Oil of chenopodium is very effective for both strongyles and cylicostomes. Animals should be fasted for 36 hours, and oil of chenopodium administered in a dose of 4 to 5 fluid drams for a 1,000-pound animal, or at a dose rate of 1 fluid dram for each 250 pounds of body weight, immediately preceded or followed by 1 quart of raw linseed oil. Cases of excessive purgation have been reported in some instances following the use of raw linseed oil. It is possible that this undesirable action is due to impurities in the product; consequently a good grade of oil should be used. Veterinarians of the U. S. Army have proposed a substitute purgative of castor oil and mineral oil, claiming that this mixture provides suitable purgation following treatment with oil of chenopodium and is without undesirable effects. The following are the doses of the mixture recommended by them: For weanlings, castor oil, 4 to 6 ounces, and mineral oil, 1 pint; for yearlings and 2-year-olds, castor oil, 6 to 8 ounces, and mineral oil, 1 pint; for 3-year-olds and older, castor oil, 8 to 10 ounces, and mineral oil, 1½ pints. It has been our experience, however, that this mixture does not provide sufficient purgation; similarly, inadequate purgation has been obtained with aloes.

Carbon tetrachlorid is very effective for the removal of large strongyles, but only about 50 per cent effective for small strongyles. Animals should be fasted for 36 hours before treatment and carbon tetrachlorid administered in a dose of 6 to 12 fluid drams for a 1,000-pound animal. Hall found carbon tetrachlorid at this dose rate to be very effective against *Strongylus* spp. De Blieck and Baudet,[13] using somewhat larger doses than those employed by Hall, confirmed Hall's results. However, Shul'ts and Raevskaia[14] reported a series of experiments on the horse on the basis of which they recommend a routine dose of 150

to 200 cc of carbon tetrachlorid for mature animals and state that the smaller doses recommended by American workers are not sufficiently effective. They conclude that at best the treatment with carbon tetrachlorid can be expected only to lower the degree of infestation and that complete elimination of the strongyles cannot be accomplished with this drug.

Wright and his collaborators recently have reported critical experiments with normal butylidene chlorid for the removal of horse parasites. The results of these tests indicate that this compound, in a dose of 0.2 cc per kilogram of body weight, is very effective for the removal of both strongyles and cylicostomes. Animals should be fasted for 36 hours before treatment. A dose of 0.2 cc per kilogram is equivalent to about 90 cc of the drug for a 1,000-pound animal. An additional test would seem to indicate that the dose of the drug may be reduced to 0.1 cc per kilogram of weight without materially reducing the efficacy of the treatment, but further tests are needed to confirm the efficacy of the drug at this dose rate. It is advisable in all cases to follow butylidene chlorid in 5 hours by raw linseed oil at a dose rate of 1 quart per 1,000 pounds of body weight.

Pinworms (*Oxyuris equi*): Oil of chenopodium, as administered for large and small strongyles, is effective for the removal of pinworms from the horse. Oil of turpentine also is an effective treatment. Animals should be fasted for 36 hours prior to treatment, and oil of turpentine administered in a dose of 2 fluid ounces for a 1,000-pound animal, immediately preceded or followed by 1 quart of raw linseed oil. Butylidene chlorid is about 50 per cent effective for the removal of pinworms.

The treatments outlined in this paper have been found by critical test to be effective for the removal of the specific parasites for which they are recommended. When used in accordance with recommendations, they should prove effective in practice. However, it must be borne in mind that no anthelmintic will remove all worms in every case. Most horses are subjected to frequent reinfestation with parasitic worms, and the arrival of the wandering larvae and agamic forms of strongyles and cylicostomes in the intestinal tract, where they develop rapidly to maturity, is more or less a continuous process. Consequently, a fecal examination two or three weeks after treatment which indicates that worms are present does not necessarily mean that the treatment was a failure. It may indicate a reinfestation which calls for additional treatment.

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Rebuilding the Royal Veterinary College

The rebuilding of the Royal Veterinary College, at Camden Town, England, is proceeding satisfactorily, according to the *Veterinary Record.* One section, including part of the canine block, the out-patients' department, and the department for dealing with the prevention of diseases in farm animals, is now completed. A gift of $25,000 has been made to endow a chair of animal husbandry, and other gifts are sought to endow chairs which are to relate to such subjects as zoölogy, botany, anatomy, milk and meat inspection, physiology, pathology, and veterinary medicine and veterinary surgery. Among the premises yet to be erected are a model dairy and a model cowshed.

The authorities hope that by 1935 the Royal Veterinary College, at Camden Town, will be an example to the rest of the world.

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STUDIES ON ULCERATIVE ENTERITIS IN QUAIL*

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Ulcerative enteritis, or "quail disease," is perhaps the disease best known and most feared by game-breeders. The increasing demand for quail for restocking purposes has resulted in an increased number of game farms and a consequent concentration of birds on a limited area. This creates a more favorable condition for the transmission of diseases in general. The devastating results following the sudden onset of an enzoötic of ulcerative enteritis under such conditions are well known to the majority of game-breeders. The yearly financial loss incurred by the mortality from this disease is unquestionably great.

GENERAL DISCUSSION

A severe epizoötic disease in grouse was brought to the attention of English sportsmen by a magazine article in 1817.[1] Various theories concerning the etiological agent of grouse disease have been advanced. However, the findings of Klein[2] and the description of the organism *Bacillus scoticus migula*, as the causative agent of the disease have been accepted generally. Such features of grouse disease as seasonal occurrence, mode of attack, and general character of the lesions strongly suggest that grouse disease and ulcerative enteritis, if not actually identical, are closely related infections. The near relationship of quail and grouse as members of the family Tetraonidae and the fact that grouse are readily susceptible to ulcerative enteritis add further support to this supposition. Morse,[3] in publishing the results of his study of "quail disease," points out the similarity of the two diseases. He isolated and incriminated in his report an organism of the colon group, which he regarded as a variant of the one described by Klein.

Morse found no protozoan organisms present in diseased birds. He states that "bacterial research gave different and far more definite results. With striking unanimity all diseased birds yielded a bacillus of the *B coli* group." He continues:

> The bacillus can not always be cultivated from the heart-blood of a bird dead with quail disease, nor can it always be cultivated from the lungs. It can usually be isolated from the liver in which lesions exist, and always from the affected intestines. Thus far in the investigation the organism has not proved pathogenic

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> for chickens, pigeons or rabbits. It has produced death in mice and guinea pigs, with the characteristic lesions.

The reader can only speculate as to the correct interpretation of this last statement, since Morse failed to explain what he considered characteristic lesions in mice and guinea pigs. Morse says further:

> With the small amount of material available we have still been successful in inoculating birds with cultures, although feeding experiments have as yet proved negative.

The latter statement clearly shows his failure to establish firmly as the etiological agent the organism described in his report. Since natural infection is contracted from the ingestion of infective material, it is apparent that the feeding test must be the criterion in attempting to incriminate any organism as the causative agent.

Pickens, DeVolt and Shillinger,[4] in the report of their investigation of ulcerative enteritis in quail, show similar results. An organism was isolated from diseased birds which proved to be pathogenic for quail and guinea pigs, but this organism failed to reproduce symptoms of the disease when fed to susceptible birds. The results of various other investigators working with this disease have been similarly conclusive in that they have failed to incriminate positively any organism as the causative agent.

Morse, on postmortem examination, found ulcerative enteritis in the common bobwhite *(Colinus virginianus)*, the California quail (*Lophortyx californicus vallicola*), the Gambel quail (*Lophortyx gambeli)*, the mountain quail *(Callipepla squamata)*, and the sharp-tailed grouse *(Pedio caetes phaseanellus campestris)*.

Levine[5] reported on a severe epizoötic of ulcerative enteritis infection in ruffed grouse *(Bonasa umbellus)*. He found that this infection in grouse was easily transmitted to quail. This widespread demonstration of ulcerative enteritis in ruffed grouse suggests the possibility of an extensive fatality in game birds from this cause.

The reports in the literature within the past twenty years have pointed out the prevalence of ulcerative enteritis. It has been reported from most sections of the United States. Much of the knowledge has been based upon field observations and inconclusive laboratory experimentation. With the increasing number of game farms, artificial propagation is rapidly becoming a factor of economic importance. It is apparent that there is a need for a better fundamental knowledge of the causative

organism and the source of infection before we can successfully prevent or attempt to treat this disease.

Material and Methods

During the course of this investigation, the bobwhite was the principal subject for experiment, although various game birds, domesticated birds, and laboratory test animals have been utilized. There have been no cases of accidental infections among the test birds.

The intestinal content from the diseased birds proved to be the best medium for transmitting the infection. The intestinal material was stripped out and diluted with 50 cc of distilled water. Saline solutions and glycerin proved to have no advantage over water for this purpose. One-tenth cc of highly virulent material would regularly reproduce the infection, but 2 cc was given into the crop as a routine practice with material of undetermined potency. The infective material was stored at room temperature, but it rapidly decreased in virulence when incubated at 37° F. or when it was held at refrigerator temperature. The organisms in the digestive contents experimented with in this investigation regularly lost their infective properties when held for more than a month.

Birds held in the laboratory were maintained on a ration of mixed grain and mash containing 1 per cent cod-liver oil. Quail receiving an increased amount of cod-liver oil appeared to be more resistant to the infection, but sufficient birds were not available to check this conclusively.

Experimental

In July, 1931, before the beginning of this investigation, a severe epizoötic of ulcerative intestinal infection in half-grown wild turkey poults was brought to our attention. These birds on necropsy presented a typical picture of ulcerative enteritis as encountered in quail and grouse. The lesions, without exception, were characteristic of this disease. The lungs were congested, small areas of superficial necrosis were present in the liver, and ulcers were found throughout the entire length of the intestines. The ulcers, which varied in size up to one centimeter in diameter, were more numerous in the posterior half of the intestines. Bacteriological and parasitological examinations were negative with the exception of a few coccidial oöcysts in one bird. This condition in no way resembled blackhead infection. The epizoötic had subsided before quail could be secured for transmission experiments.

The following year, two wild turkey poults were obtained for tests. These birds were each fed a small amount of intestinal content from a quail with ulcerative enteritis. Both birds developed a diarrhea, and one died on the fifth day of the test. The lesions found in this bird on necropsy were suggestive of the infection. Hemorrhagic areas covered with a croupous exudate were found throughout the intestines. The lesions in the duodenum were more numerous and more closely resembled true ulcer formations. Cultures made from the internal organs proved to be negative. Intestinal material from this bird, however, was non-infective for quail. The remaining bird appeared to be normal when it was killed for examination two weeks later.

In June, 1932, five one-month-old chicks were examined from a flock in which 750 had died within three days following a week of cool and rainy weather. The owner had opened a number of diseased birds and observed that the intestines were filled with a cheesy material. Characteristic lesions of ulcerative enteritis were present in three of the five chicks examined. Cultures made from the internal organs were negative. Intestinal content from these chicks was fed to five healthy chicks of the same age. One chick died a week later. The lesions consisted of congestion of the liver and catarrhal enteritis.

Two quail were given small amounts of the intestinal contents from the infected chicks. A marked diarrhea was apparent on the third day. The quail died on the fifth and seventh days of the test and characteristic ulcerations of ulcerative enteritis were found in these birds at necropsy. The intestinal content from these quail did not induce infection when fed to healthy quail or chicks.

Somewhat similar results were obtained on a later test. Intestinal content from a half-grown chicken with intestinal ulcers was fed to two quail. One of the quail died on the ninth day of the test with ulcerative enteritis, while the other remained healthy. The intestinal material from both the chicken and the quail proved to be non-infective on repeated tests.

No definite conclusions can be drawn from these experiments. The chickens used in the transmission experiments were apparently highly resistant to the infection. It is doubtful whether the one chicken that died during the transmission tests was infected with ulcerative enteritis, since there were no true ulcer formations in the intestines. The intestinal content from the chickens with lesions characteristic of ulcerative enteritis was, without doubt, infective for quail. This suggested the possibility of apparently normal chickens acting as carriers of the infection.

Natural Resistance

The degree of resistance in quail to the infection varies and inconsistent results often are obtained by feeding infective material to apparently susceptible birds. Infection in some birds appears to build up an active immunity of considerable duration. Three birds, survivors of three affected groups of 15 birds each, have demonstrated an immunity to subsequent infections when tested repeatedly with infective material of known virulence. These three birds manifested symptoms of dullness, diarrhea, and a rapid loss of weight. The presence of the primary infection in these birds was established by feeding their droppings to susceptible birds.

The average period of incubation appears to be about four days. There is, however, considerable variation in this time. Infectious material secured from one source consistently produced death 48 hours after feeding, while material from another source induced chronic infection, with an incubation period of two weeks or longer. The observations are further complicated by the fact that infectious material, when fed simultaneously to two similar birds, may induce chronic infection in one and acute infection in the other. Continual passage from one bird to another, maintained in laboratory environment, in every instance tended to decrease rather than increase the virulence of the infectious material. All attempts to increase the virulence of the infective material under natural conditions have failed.

Feeding Tests

Natural infection could not be induced either in the laboratory or under game-farm conditions when the birds were overcrowded in insanitary quarters. Macerated liver, lungs, spleen, heart and heart-blood have failed to transmit the infection, thus far, when given per os. The tissues for these feeding tests were selected from birds which had no perforating ulcers for the puurpose of eliminating the possibility of surface contamination of the internal organs, which might result from ulcer perforation. Feeding of the lesions of blackhead infections from quail and turkeys has failed consistently to produce ulcerative enteritis in quail and grouse.

Infectious material of known virulence for quail has failed to reproduce this infection when repeatedly fed to pigeons, rabbits, guinea pigs, white rats, and mice. Negative results also were obtained by feeding internal organs from diseased birds.

Morse[3] reported that the organism which he isolated from

diseased birds would produce death in mice and guinea pigs with characteristic lesions. If by "characteristic lesions" he meant an intestinal infection with the typical ulcerations, it is logical to assume that similar results could be obtained by feeding virulent infective material.

Protozoan Organisms

Gallagher[6] suggests the possibility of coccidia as a factor in the formation of the lesions. Coccidial oöcysts may be present in the intestines of birds dead with the disease, but this is an exception rather than the rule. Histopathological study of the intestines from birds dead 48 hours after the ingestion of infectious material does not reveal any lesions that could be attributed to coccidia. Coccidial lesions have been found but not in association with the ulcerations. The average period in quail existing between the ingestion of infective oöcysts and the passage of the non-sporulated forms in the feces is about four days. Considering these facts it is hardly conceivable that coccidia would cause death in 48 hours or play any part in the formation of the lesions. If coccidia were a factor in the disease, the intestinal content would not be infective until the oöcysts had sufficient time to sporulate.

No protozoan organisms, other than coccidia, have been observed regularly in cases of ulcerative enteritis. Certain forms which in some respects resembled an encysted flagellated organism were found in a few birds. Attempts were made to cultivate these forms on egg media. Feeding tests made with these cultures were negative.

A study of the blood-pictures from a number of infected birds did not reveal any parasitic organisms. An incubation period of only 48 hours, in general, would eliminate most of the protozoan organisms as the probable etiological agent. According to an oral communication with Dr. Justin Andrews, protozoölogist at Johns Hopkins University, no protozoan organisms were found in the diseased birds examined by him.

Bacteriological Results

An adequate discussion of this phase of the investigation would require a lengthy paper on this subject alone. The bacteriological study of the disease gave results entirely contrary to those obtained by Morse. Culturing of the spleen, liver and blood gave consistently negative results. Contaminating organisms are sometimes cultivated from the internal organs following postmortem changes in the birds. There were a variety of con-

taminating organisms found, none of which was pathogenic for quail when given per os. No growth was obtained on different media by varying the pH or temperature ranges.

As a rule, cultures grown from the intestines were noninfective on feeding tests. Anaerobic organisms thus far have given negative results. What appear to have been typical cases of the disease have been transmitted to groups of two birds each on three different occasions by feeding mixed aerobic cultures obtained from intestinal ulcers. Subsequent trials with transfers from these infective cultures gave no results. The intestinal content from these infected birds did not reproduce the disease when fed to other groups. Pure cultures of the organisms separated from the mixed infective cultures proved to be nonpathogenic on feeding tests. Attempts to increase the virulence by the usual methods were unsuccessful.

The results obtained in the above experiments were carefully checked under controlled conditions. While it appears that some aerobic organism is responsible for the infection, it rapidly loses its pathogenic powers on culture media.

Tests were made to determine if the infective agent of the disease is a filter-passing virus. Tissue extracts of the liver and heart-blood were passed through Berkefeld and Chamberland filters. The bacteria-free filtrate was fed and injected into quail, pigeons, chickens, rabbits, guinea pigs and rats. Suspension of the residue from the filtration also was fed to identical groups. Both of these experiments gave negative results.

A solution of infective intestinal content was passed through Chamberland and Berkefeld filters and fed and injected into similar test groups with no results. The unfiltered portion washed from the filter candles reproduced the disease in quail. Identical results were obtained on repeated tests.

According to a letter from Dr. Justin Andrews, similar results were obtained by passing infective intestinal content through a double Seitz disc.

Death of birds in the acute stage of the disease before sufficient time has elapsed for lesions to develop suggests the possibility of a potent toxin-producing organism as the causative agent. This is supported by the study of the lesions in the internal organs. The experiments previously mentioned eliminate the probability of a filtrable virus as the etiological agent. They also demonstrate the absence of toxins in the tissue extracts and intestinal contents in sufficient amounts to produce either death or symptoms of the disease.

The disease often assumes a chronic course, in which cases perforating ulcers are sometimes found in the intestines. Resulting peritonitis is the logical explanation for the cause of death in these birds. Cultures made from the peritoneal fluid and serous surface of the intestines of these birds are thus far consistently negative.

Pickens, DeVolt and Shillinger[4] isolated a pathogenic organism by injecting tissue extracts of the liver from diseased birds into guinea pigs. This organism was pathogenic when injected into birds, but gave negative results on feeding tests. Since it has been demonstrated in our investigations that quail will succumb following an injection into the body cavity of such common organisms as *Escherichia coli*, their observations carry no great significance. In view of the facts that cultures of the liver are usually negative and that liver tissue apparently does not carry the infection as shown by feeding tests, it appears probable that they were working with contaminating organisms. The areas of necrosis usually found in the liver offer favorable conditions for the multiplication of any contaminating organisms entering through the intestinal opening of the bile-duct. The disease has not been produced by subcutaneous or intramuscular injections of the infective material or organisms cultured from the intestines. The infection is evidently not contracted through any portal other than the alimentary tract.

On a very recent occasion a micrococcus, which has not been described, was isolated in pure culture from the peritoneal cavity of a bird with ulcerative enteritis. No conclusive tests with this organism have as yet been made.

SYMPTOMS

The average period of incubation, as previously stated, appears to be about four days. Individuals affected with the acute forms of the disease frequently die without showing symptoms. Affected groups usually show a diarrhea which is made evident by white spots on the drop-boards or ground of the pen.

Birds chronically affected appear dull and listless, with feathers ruffled. Extreme emaciation is noted, the birds sometimes losing from a third to a half of their original weight.

PATHOLOGY

The gross pathologic changes of ulcerative enteritis are confined to the intestines, lungs and liver. The intestines usually are found to be studded with small ulcers, some of which may have perforated the intestinal wall. Often there are caseous

casts in the ulcers. The duodenum sometimes is enlarged considerably and reddish purple in color. Rarely the birds show any intestinal lesions other than definite areas of hemorrhagic enteritis. The lungs are congested regularly and exhibit areas of consolidation occasionally. The liver does not always contain necrotic areas on gross examination, but histopathologic study demonstrates extensive degeneration and extreme congestion.

Discussion

With many unusual and seemingly contradictory results obtained in the study of ulcerative enteritis in quail and grouse, it is difficult to draw any conclusions relative to the nature of the etiological agent. Our inability to develop or maintain strains of the organism of a high virulence under controlled conditions is baffling in the face of the often disastrous spread of the infection in flocks where it becomes epizoötic. The violence of outbreaks and the difficulty of controlling the spread are well known. Instances are recorded where 75 per cent of flocks succumbed in brief periods.

It is possible that our understanding of the susceptibility of birds is incorrect. Evidence indicates that birds on a ration low in certain vitamins may become infected much more readily. It has been observed that some of the most disastrous outbreaks of ulcerative enteritis have occurred in flocks widely removed from their natural environmental conditions. The disease has appeared with equal violence in Mexican quail imported for stocking purposes and native quail and grouse in various parts of this country. Outbreaks appear to have begun spontaneously in old established flocks with no introduction of a new stock of game birds. The possibility, however, of its introduction by wild birds of various species has not been eliminated in these instances.

The only known control measures at present are those of breaking up flocks into as small groups as possible in widely separated locations and preventing spread of the infection by the usual hygienic procedures.

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[2]Klein: The grouse. Health and Disease (London, 1911), ii.

[3]Morse, G. B.: Quail disease in the United States. U. S. Dept. of Agr., B. A. I. Cir. 109.

[4]Pickens, E. M., DeVolt, H. M., and Shillinger, J. E.: An outbreak of quail disease in bob-white quail. Maryland Conservationist, Spring issue (1932), p. 18.

[5]Levine, P. P.: A report on an epidemic disease in ruffed grouse. Trans. 19th Amer. Game Conf. (1932), p. 437.

[6]Gallagher, B. A.: Amer. Game Pro. Asso. Bul. (Apr., 1924), pp. 14-15.

DISCUSSION

DR. SHILLINGER: My observations have been very inconclusive. The most prominent features of the disease that we have encountered have been the violent outbreaks on game-farms and in flocks of birds that were shipped in concentrated numbers for restocking purposes, and the contradictory and baffling experience we have had in carrying on our experiments. That is, the species of infected birds have prevented the usual dogmatic manner of carrying on laboratory tests with infected material.

Unfortunately, so far we have not incriminated any one particular organism, but as stated in the paper, in many instances under laboratory conditions the infectious agent appears to lose virulence. There is a strong suspicion on my part that the susceptibility of the birds depends greatly on the manner in which they may be fed or handled prior to the outbreak of the disease, but the disease is very distinct in its characteristics, and causes very typical symptoms.

DR. R. FENSTERMACHER: In the diagnosis laboratory this past season, I received two or three lots of young turkeys which had lesions containing the same condition described by Dr. Shillinger, and a large percentage of them died. It was a native group of turkeys and out of several thousand we would lose 50 to 100 a day for a while, and then it would stop just as quickly as it started.

DR. SHILLINGER: In domestic birds, geese, ducks and turkeys, in which we have seen this disease, or found what appeared to be a similar condition, the turkey has been the most frequently infected. The wild birds may be more or less natural reservoirs for the disease, but it seldom manifests itself as a severe outbreak such as occurs in quail and grouse.

DR. ROBERT GRAHAM: Dr. L. P. Doyle, of Purdue University, has found ulcerative enteritis in a number of flocks of young turkeys this year, and he feels that this is or is becoming possibly a very important disease of young turkeys. I believe in a few years we will hear a great deal more about ulcerative enteritis.

DR. E. M. DICKINSON: We have encountered this same condition for the last couple of years, and there is no question but that it is becoming a very serious problem in our turkey industry. As far as getting any information is concerned, we are getting all we possibly can. You know we are in a section that is rather extensively engaged in producing turkeys, and we find this disease occurs in some instances where our flocks are maintained on wire porches, which is something rather unusual, because very often wild birds are more or less eliminated from associating with our birds, that is, perhaps, with the exception of sparrows or small birds of that nature. Very often our birds will have a covering over their porches outside in order to keep them separate from the wild birds.

DR. SHILLINGER: The most disastrous outbreak we have had was on one of the eastern state game-farms during the winter months when they were carrying over some 1,800 birds. A large portion of that flock—I believe it was very nearly 50 per cent of the mature birds—died. They were maintained in wire cages, about 30 to a cage. There was a wire-mesh floor in the coops in which they were kept at night, each coop having about one foot of straw. This straw was changed rather frequently and did not give the appearance of a highly polluted bedding. Of course, there was some accumulation of feces there.

DR. HUGH HURST: I would like to know if there is any treatment that has proved satisfactory in eliminating this disease from the flock after it is once affected.

DR. SHILLINGER: I am sorry to say there is nothing that we have found to be satisfactory with quail. On some of these farms the caretakers are not unlike the caretakers of domestic poultry and other farm stock. They have tried nearly everything possible, and the disease seems to continue just as bad as when it started, despite any treatment. I believe that isolation, quarantine and the breaking up of the flocks into small groups are the best known methods of checking the disease.

DR. DICKINSON: Have you found any apparent age difference in susceptibility? We find that after turkeys are twelve weeks of age they are not so susceptible. The mortality is greatly reduced at about three or four months of age. From then on the mortality seems to slow down and we don't have the severe infection.

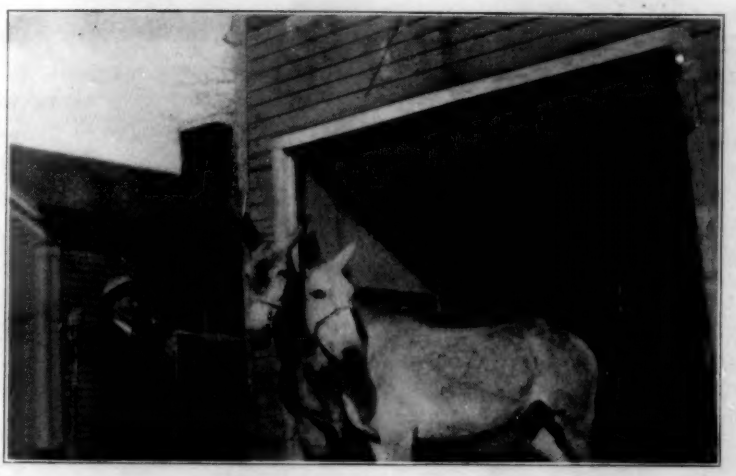
DR. SHILLINGER: With quail and grouse, I don't believe that holds. Without records here to refer to, I believe the fully adult birds are somewhat more resistant, that is, birds a year old or older.

Toil, says the proverb, is the sire of fame.—EURIPIDES.

12th International Veterinary Congress
New York—August 13-18, 1934

From Garage to Sales Stable

Dr. Fred H. Steele, of Huntsville, Ala., submitted the accompanying photograph of a sales stable that, previous to alterations, had been a garage. Dr. Steele offered apologies to Ripley (Believe It or Not) and John Hix (Strange as It May Seem). We all know scores of instances of stables and barns having been converted into garages, but this is the first case of the reverse order that has been brought to the attention of the JOURNAL.



A SALES STABLE THAT WAS A GARAGE

ACUTE ENTERITIS IN YOUNG PIGS DUE TO INFECTION WITH COLON GROUP*

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INTRODUCTORY

The investigations reported in this paper were made at a large garbage-feeding ranch in southern California early in the spring of 1933. About 75,000 to 80,000 pigs are farrowed annually on this ranch.

An unusually heavy loss in newborn pigs began to attract the attention of the ranch management in the early part of November, 1932. The losses increased during November and December. There was a slight recession during January, but the mortality then mounted again and was very heavy during February, March and April, and became a matter of serious concern to the ranch management, as the production of baby pigs was reduced to a serious and alarming extent. The losses ranged from 30 to 60 per cent for the entire ranch and in one of the brooder units ran as high as 70 to 80 per cent for several weeks.

At first it was thought that the trouble might be due to some nutritional cause or mineral deficiency and the ranch veterinarian had carried out a considerable amount of experimental work along these lines without achieving much success in checking the losses.

Experiments also had been carried out in the hope of eliminating the trouble through improved sanitation in the brooder units. Thus, several "batteries," each consisting of 25 farrowing-pens, and one individual unit, consisting of 100 farrowing-pens, were scrupulously cleaned and thoroughly disinfected, but there was little, if any diminution in the losses when sows were allowed to farrow in these pens.

This experimental work, while it yielded mostly negative results, was nevertheless of value in eliminating from the problem possible factors which needed to be taken into consideration.

For carrying out the present investigations, a temporary bacteriological laboratory was set up at the ranch, with a limited amount of laboratory equipment. Space was assigned in a building equipped with gas, electric current and running water, which answered fairly well for bacteriological work.

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*Presented at the seventieth annual meeting of the American Veterinary Medical Association, Chicago, Ill., August 14-18, 1933.

Description of the Disease

The disease was found to prevail in newborn pigs in the farrowing-pens and was characterized by a heavy mortality within two to five days after birth. The disease came to be designated by ranch attendants as "the little-pig disease" or the "three-day-pig disease" from the fact that death occurred so frequently at this age.

The chief clinical manifestations of the disease were weakness, unsteady gait, emaciation, roughness of hair, and often a wrinkled appearance of the skin. In the typical cases, the little pigs presented a droopy and listless appearance, standing with their heads lowered and noses almost touching the ground in a state of apparent stupor. About 50 per cent of the affected pigs showed a yellowish diarrhea or scours. (The characteristic appearance of a little pig affected with the disease is well shown in figure 1.)

Fig. 1. The characteristic appearance of a pig affected with acute enteritis.

In the typical cases, the disease presented the characteristics of an acute and rapidly invading infection, developing shortly after birth. A number of pigs in a litter were apt to be affected and sometimes entire litters succumbed. Healthy litters and badly affected litters frequently were observed in adjoining farrowing-pens, indicating that the infection was not of a contagious nature.

Upon postmortem examination, it became quite evident that a large number of little pigs were dying as a result of a severe enteritis, involving the small intestine and confined chiefly to the lower duodenum and jejunum.

The enteritis was very acute and severe in the typical cases, more so, apparently, than that seen in older pigs. In some cases, which were evidently very early ones, the enteritis was less severe, while in still other cases it had passed the acute stage and had reached the stage of necrosis. In some cases it involved the greater portion of the small intestine, but was more often confined to a more limited area of a few feet. In the typical acute cases, the affected portion of the intestine was intensely hyperemic and presented a crinkled, bubbly appearance, indicating gas formation in the wall of the intestine. The entire intestinal wall often presented a dark red appearance.

Aside from small, pin-point petechiae in the kidneys, which seem to have little pathological significance in baby pigs, there were no noteworthy lesions in the internal organs. The stomach and stomach contents appeared normal, the stomach being filled, as a rule, with well-formed curd, indicating that the little pigs had been nursing well. In the early cases, the mesenteric capillaries were apt to be congested and in typical acute cases the mesenteric glands often were enlarged and congested.

From clinical observations made in the farrowing houses, it became obvious that a large number of weak litters were being farrowed. As a consequence, a large number of weak, runty pigs were being killed by the brooder attendants immediately after birth. All pigs which died a day or more after birth were collected and autopsied by the ranch veterinarian. Of the pigs that came to autopsy, somewhat more than 50 per cent showed intestinal lesions. It seemed rather evident, therefore, that some factor was present in the herd responsible for the birth of weak pigs and that about 50 per cent of these pigs were dying from an enteritis, probably due to a secondary infection.

Similar conditions were reported from other garbage-feeding ranches in southern California and several of these had been visited by the ranch veterinarian, who found the same disease was prevailing on other ranches. It was reported that one of these ranches had lost 90 per cent of the entire pig crop, which, of course, was a ruinous loss, if true. It also was reported from these ranches that the disease had existed for a time and then disappeared spontaneously, but the truth of these statements could not be verified.

Bacteriological Studies

Pigs were selected for bacteriological examination which presented typical clinical symptoms of the disease and were usually

taken from litters in which several were affected and there had been some death loss.

Most of the pigs were brought to the laboratory alive, killed by a blow on the head and cultures secured immediately after opening the carcass. A few which had died very recently and were perfectly fresh also were cultured.

Groups of pigs were selected for examination in conformity with a classification that had been adopted by the ranch veterinarian in recording postmortem examinations, as follows:

1. *Suspicious:* So classed on general postmortem appearance, emaciation, rough hair, etc. Pigs in this group sometimes exhibited some reddening or hyperemia of the intestinal wall, but often the intestines were quite normal in appearance.
2. *Cases:* In this group, there was well-marked reddening or hyperemia of the small intestine and these pigs might be regarded as exhibiting an early stage of an acute enteritis.
3. *Typical:* These were cases in which there was a severe enteritis, with intense reddening or hyperemia of the intestinal wall, which often presented a crinkled or bubbly appearance, indicating gas formation.
4. *Chronic Cases:* These were cases of somewhat longer standing (6 to 12 days), in which there was evidence of necrosis of the intestinal mucosa.

A total of 31 pigs were cultured, which were classified as follows: 8 suspicious, 6 cases, 11 typical, and 6 chronic cases.

Cultures were made, as a rule, from the heart-blood, spleen, liver and kidney, and from the mesenteric glands when these were enlarged and congested. In the group of typical cases, Endo's plates also were made from the contents of the small intestine, in the affected areas. Cultures were not made from all the organs in every instance, owing to the limited supply of laboratory glassware and the consequent limitation of culture media available at any one time. Sufficient cultures were made from each animal, however, to determine whether bacterial invasion had occurred.

Because of facility of preparation and lack of laboratory equipment and assistance, Difco media were used, including nutrient agar, North gelatin agar, Endo's agar, Russell double-sugar agar, nutrient broth, litmus milk, phenol red dextrose broth, phenol red lactose broth, and phenol red saccharose broth.

The results of bacteriological studies are shown in tables I to IV.

Discussion of Bacteriological Results

A study of the tables reveals an extensive and striking invasion by *Bacillus coli* in all four groups. In the three groups of pigs classed as "cases," "typical" and "chronic cases"—that is, in all of the pigs cultured except the very early cases—in-

TABLE I—*Group 1. Pigs classed as "suspicious." (Bacteriological findings showed invasion by B. coli in 75 per cent.)*

PIG	AGE (DAYS)	AUTOPSY FINDINGS	BACTERIOLOGICAL FINDINGS			
			HEART-BLOOD	LIVER	SPLEEN	KIDNEY
1	3	Kidneys pale with numerous pin-point petechiae; other organs normal. Acute enteritis involving lower duodenum and jejunum	No growth	No growth	No growth	No growth
2	4	Kidneys pale with small pin-point petechiae. Other organs normal. Mucous membrane of lower duodenum and jejunum swollen, but not noticeably reddened. Mesenteric capillaries congested	No growth	No growth	.....	*B. coli*
3	4	Kidneys showed small pin-point petechiae. Other organs normal. Beginning enteritis involving lower duodenum and jejunum. Mesenteric capillaries congested	*B. coli*	.....	No growth	*B. coli*
4	5	Kidneys pale with small pin-point petechiae. Other organs normal. Mucous membrane of lower duodenum and jejunum swollen, but not noticeably reddened. Mesenteric capillaries congested	Gram-positive coccus	.....	.....	*B. coli*
5	4	Kidneys showed small pin-point petechiae. Other organs normal. Beginning enteritis involving lower duodenum and jejunum. Mesenteric capillaries congested	*B. coli*	No growth	.....	.....
6	6	Same as pig 5	No growth	No growth	No growth	.....
7	4	Same as pig 5	No growth	*B. coli*	No growth	*B. coli*
8	6	Same as pig 5	*B. coli*	.....	*B. coli*	.....

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TABLE II—*Group 2. Pigs classed as "cases." (Bacteriological findings show invasion by B. coli in 100 per cent.)*

PIG	AGE (DAYS)	AUTOPSY FINDINGS	BACTERIOLOGICAL FINDINGS			
			HEART-BLOOD	LIVER	SPLEEN	KIDNEY
1	3	Kidneys pale with numerous pin-point petechiae. Other organs normal. Early enteritis involving greater portion of duodenum and jejunum	*B. coli*	No growth	No growth	*B. coli*
2	1	Liver and kidneys pale; latter showed pin-point petechiae. Other organs normal. Early enteritis involving lower duodenum	*B. coli*	No growth	No growth	*B. coli*
3	3	Spleen slightly congested. Other organs normal. Acute enteritis involving lower duodenum and jejunum	No growth	No growth	.....	*B. coli*
4	3	Lungs pleuritic adhesions. Liver and kidneys pale; latter showed pin-point petechiae. Enteritis involving lower duodenum; intestinal folds matted together	*B. coli*	.....	*B. coli*	.....
5	5	Lungs normal. Liver, spleen and kidneys somewhat congested. Acute enteritis involving greater portion of small intestine	*B. coli*	.....	*B. coli*	.....
6	12	Lungs revealed several areas of pneumonia. Spleen enlarged and congested. Kidneys showed small petechiae. Acute enteritis involving lower duodenum and jejunum	.....	.....	*B. coli*	.....

TABLE III—*Group 3. Pigs classed as "typical." (Bacteriological findings showed invasion by B. coli in 100 per cent.)*

Pig	Age (Days)	Autopsy Findings	Bacteriological Findings					
			Heart-Blood	Liver	Spleen	Kidney	Mesenteric Gland	Small Intestine
1	4	Spleen some enlarged and slightly engorged; other organs normal. Acute enteritis with beginning necrosis involving lower duodenum; intestinal folds matted together	No growth	*B. coli*	*B. coli*	*B. coli*	.....	*B. coli*
2	5	Kidneys pale with numerous pin-point petechiae; other organs normal. Acute enteritis with beginning necrosis involving lower duodenum	*B. coli*	*B. coli*	.....	*B. coli*	.....	*B. coli*
3	5	Same as pig 2	*B. coli*	No growth	.....	No growth	.....	*B. coli*
4	3	Kidneys pale with numerous small petechiae; other organs normal. Acute enteritis involving greater part of small intestine and also lower intestine	*B. coli*	.....	*B. coli*	No growth	.....	*B. coli*
5	3	Same as pig 4	No growth	.....	*B. coli*	*B. coli*	.....	*B. coli*
6	3	Kidneys pale with numerous petechiae; other organs normal. Acute enteritis involving practically whole small intestine and large intestine. Mesenteric glands intensely congested	*B. coli*	*B. coli*	*B. coli*	No growth	*B. coli*	*B. coli*
7	4	Spleen slightly engorged; kidneys pale with numerous small petechiae. Acute enteritis involving lower duodenum and jejunum. Mesenteric glands enlarged and intensely congested	*B. coli*	*B. coli*	*B. coli*	No growth	*B. coli*	*B. coli*
8	3	Kidneys pale with numerous small petechiae; other organs normal. Acute enteritis involving greater part of small intestine and also large intestine	No growth	No growth	Lactobacillus	.....	*B. coli*	*B. coli*
9	5	Spleen and kidneys slightly congested. Acute enteritis involving greater part of small intestine. Mesenteric glands congested	No growth	.....	*B. coli*	*B. coli*	*B. coli*	.....
10	7	Same as pig 9	No growth	.....	*B. coli*	.....	.....	.....
11	4	Same as pig 9	*B. coli*	.....	*B. coli*	.....	*B. coli*	.....

TABLE IV—*Group 4. Pigs classed as "chronic cases." (Bacteriological findings showed invasion by B. coli in 100 per cent.)*

Pig	Age (Days)	Autopsy Findings	Bacteriological Findings				
			Heart-Blood	Liver	Spleen	Kidney	Mesenteric Gland
1	5	Spleen slightly congested; other organs normal. Necrotic enteritis involving lower duodenum and jejunum	No growth	.....	*B. coli*	*B. coli*	.....
2	5	Kidneys showed small pin-point petechiae; other organs normal. Small intestine showed acute enteritis in upper portion, tending to become necrotic in lower portion	No growth	No growth	No growth	.....	*B. coli*
3	6	Spleen slightly congested; other organs normal. Enteritis involving lower duodenum, tending to become necrotic	No growth	.....	*B. coli*	.....	.....
4	6	Spleen somewhat congested; other organs normal. Necrotic enteritis involving lower two-thirds of small intestine	*B. coli*	.....	*B. coli*	.....	.....
5	9	Spleen, liver and kidneys somewhat congested. Necrotic enteritis involving lower portion of small intestine	.....	.....	*B. coli*	.....	*B. coli*
6	12	Spleen and kidneys somewhat congested; other organs normal. Necrotic enteritis involving lower duodenum and jejunum	.....	.....	*B. coli*	.....	*B. coli*

vasion by *B. coli* occurred in 100 per cent of these pigs. In the group classed as "suspicious," which would represent very early cases, invasion by the colon organism was noted in 75 per cent of the cases.

Further study of the tables reveals the fact that *B. coli* was isolated from all of the cultures obtained from the mesenteric glands, from 72 per cent of the cultures taken from the kidney, from 68 per cent of the cultures from the spleen, and in 45 per cent of the cultures taken from the heart-blood. After invasion through the intestinal wall, it seems probable that the organism locates first in the mesenteric glands, which would explain why it was found in all of the cultures taken from these glands. The presence of *B. coli* in 45 per cent of the heart-blood cultures would indicate a generalized invasion by the organism in these cases and shows very clearly the markedly invasive properties which this organism may assume at times.

In addition to *B. coli*, a Gram-positive coccus was isolated in a few instances and an occasional streptococcus, but these organisms were not encountered often enough to be of any pathological significance. From the bacteriological studies, it seemed quite evident, therefore, that *B. coli* was the invading organism responsible for the enteritis.

Further confirmation of the bacteriological studies is afforded by microscopic sections of intestines, prepared by Dr. Karl F. Meyer, of the University of California, who visited the ranch when the investigation was in progress. Through the ranch veterinarian, Dr. Meyer obtained portions of small intestine from pigs showing the disease and from these specimens sections were prepared and stained with Giemsa solution. Through the courtesy of Dr. Meyer, opportunity was afforded to examine these sections. They revealed an extensive and destructive invasion of the mucosa by organisms which apparently corresponded in morphology with *B. coli*. The histologic picture, as described by Dr. Meyer,[1] was as follows:

> The mucous lining and part of the submucosa are completely necrotic; in fact, the villi are merely shadows. The coagulation necrosis is mixed with fibrin, and the dead fragments of tissue are covered with millions of bacteria in the areas not as yet necrotic. The principal foci of multiplication of the bacteria which resemble the colon bacillus are in the crypts. Attention is called also to the hemorrhagic edema and the gas bubbles in the submucosa all along the intestinal canal. The defensive response is slight. As a whole, the histologic picture is that of an enteritis diphtheroides.

The disease in little pigs seems to resemble rather closely the early diarrhea or "scours" of young calves, investigated by Smith

and Orcutt[2] and found by them to be due to infection by colon organisms. These investigators found that in newborn calves not sucking bacteria penetrated as far as the cecum within 12 hours. They attributed this to the fact that the meconium is liquid and favorable to the rapid diffusion of bacteria as far as the cecum, while below this it becomes more consistent and much drier and therefore less favorable for bacterial growth. They reached the conclusion that the disease was associated with the multiplication of special strains or races of *B. coli* and that such races are developed and maintained in large herds. Carrying this conclusion further, they state:

> Each large herd, through the presence of calves below par at birth, may thus develop and maintain its own type of scours organism which, however, is not virulent enough to make any headway in naturally strong calves properly cared for as regards food and housing.

They also refer to inherited morphological defects of the digestive tract in explanation of why certain calves were below par. The first 48 hours was found to be the critical period in the life of the young calf and they considered the calf to be doomed if the colon organism multiplied freely during this period. Furthermore, they found that calves quickly acquired a high degree of resistance to *B. coli* entering the digestive tract after the first week. In referring to the strains of *B. coli* isolated by them, these authors state that at the beginning of their investigation the dominant race was saccharolytic and motile, whereas, later on, this type was supplanted by one that was non-saccharolytic and non-motile.

No systematic study was made of the colon types or races isolated from the young pigs in the present investigation. Cultural and biological differences were noted, however, in that some strains fermented saccharose while others did not and some were motile while others were not. The predominant strain was saccharolytic and motile.

It is believed that conditions similar to those just noted for calf scours obtained in the swine herd under investigation and it seems probable that this herd had developed a race or strain of *B. coli* extremely virulent for little pigs below par or with a lowered resistance, but not of sufficient virulence to cause the death of the stronger pigs. It is believed that infection occurs by way of the mouth, as in the case of calf scours, and is acquired from colon organisms on the sows' udders or in the teat canals, which are taken in with the colostrum at the first nursings.

A preliminary report of an investigation of a disease in young lambs, which prevailed on widely separated farms in California,

recently has been made by H. H. Heller,[3] who found this disease to be caused by *B. coli* and it would seem to have points in common with the little-pig disease described in the present paper.

While the bacteriological studies and the histologic findings in the case of the little-pig disease seemed to indicate quite clearly that *B. coli* was the offending organism responsible for the enteritis, it seemed quite evident that some other factor or factors were present in the herd responsible for the low resistance of large numbers of the little pigs, for it seems to be quite generally conceded that *B. coli* is usually non-pathogenic and does not assume disease-producing properties or become invasive unless there is a lowering of resistance in the host animal.

Brucella Infection as a Factor

Brucellosis had been considered from the first as a possible factor in the little-pig disease and as soon as the bacteriological studies seemed to incriminate *B. coli* as the offending or causative organism in the enteritis, more serious attention was given to the possibility that Brucella infection in the herd might be the primary factor responsible for the farrowing of weak litters and the consequent lowered resistance of many of the little pigs.

Brucella antigen was secured from several sources and agglutination tests were begun with blood samples taken from sows which had farrowed weak pigs and lost part of their litters. Tests at first were made by both tube and plate methods, but the plate method was soon adopted as a routine procedure because of its simplicity and rapidity. Blood samples, taken by the ranch veterinarian and his assistants, were brought to the laboratory and the agglutination tests were made as rapidly as possible. The first series of tests on 76 blood samples yielded the results shown in table V.

While the above tests did not include a large number of animals, these tests nevertheless are regarded as sufficient in number to afford a representative cross-section of conditions obtaining on the ranch relative to Brucella infection and the fact that 73.3 per cent of the sows tested and 32 per cent of the boars gave positive agglutination tests served to indicate the widespread prevalence of brucellosis on the ranch at the time these tests were made. From these findings, it is believed that Brucella infection is a serious problem at the ranch and probably is a primary factor in the production of weak pigs with lowered resistance, which are thus rendered susceptible to

invasion by *B. coli*. The presence of brucellosis in the herd would explain the presence of weak litters affected with enteritis in farrowing-pens adjoining pens containing healthy litters entirely free from enteritis.

The investigations of Haring, Traum, Hayes and coworkers, of the Division of Veterinary Science of the University of California, would seem to indicate that Brucella infection is gaining a considerable foothold in the swine herds of California. Outbreaks of the disease were reported by Hayes and Traum[4] as early as 1920, and studies of several other outbreaks were reported by Hayes and Phipps,[5] in 1921. In a study of brucellosis in the swine herd of the University of California in 1930, How-

TABLE V—*Results of tests of 76 blood samples.*

TYPE OF ANIMAL	ANIMALS TESTED	RESULTS OF AGGLUTINATION TESTS (PLATE METHOD)			
		NEGATIVE	SUSPICIOUS	POSITIVE	
				No.	%
Sows.....	45	11	1	33	73.3
Boars.....	25	16	1	8	32.0
Gilts.....	6	4	1	1	16.6

arth and Hayes[6] found that the disease had existed in this herd in enzoötic form for many years.

Consideration of the reports just cited in conjunction with mortality records for the herd under investigation, over a considerable period of years, makes it appear certain that Brucella infection has existed for some years in this herd and has been increasing gradually. As a result, increasing numbers of weak pigs below par were farrowed, which facilitated the development of a virulent colon race, with consequent heavy losses from enteritis. A culmination of these untoward factors or influences would seem to explain the marked increase in mortality of newborn pigs, which attracted the attention of the ranch management in November, 1932, and later provided the motive for the present investigation.

From observation of the breeding methods which were being followed at the ranch, it is believed that long-continued inbreeding also was a factor in the production of weak pigs at this ranch, but it would appear, from the history of the disease at

other ranches, that Brucella infection was probably the chief primary factor in the little-pig disease described in this paper.

While extensive losses in calves and young lambs through infection by *B. coli* have been reported (*loc. cit.*), it is believed that this is the first time that a heavy loss in little pigs from infection by this organism has been reported.

SUMMARY

The investigation reported in this paper was carried out at a large garbage-feeding ranch in southern California, where there was a heavy mortality in little pigs from two to five days old.

The clinical symptoms of the disease were weakness, characterized by unsteady gait and disinclination to move, roughness of hair, emaciation and wrinkled appearance of the skin. In typical cases, the little pigs presented a droopy and listless appearance, standing with their heads lowered and noses almost touching the ground in a state of apparent stupor. About fifty per cent showed scours.

Postmortem examination revealed a severe enteritis, confined chiefly to the lower duodenum and jejunum, with no noteworthy lesions in the internal organs. In some cases, the greater portion of the small intestine was involved, but more often a section of from one to three feet was affected. The enteritis was of a more acute and severe type than that seen in older pigs on the farm. The entire intestinal wall was intensely hyperemic, of a deep or dark red color, and the tube often presented a crinkled or bubbly appearance, indicating gas formation within the coats of the intestinal wall.

A bacteriological study was made of 31 selected cases, representing very early cases, acute cases, and cases in which the disease had progressed to a more chronic stage, characterized by beginning necrosis. In every case in which enteritis had developed, cultures showed invasion by *B. coli*. The organism was recovered quite consistently in cultures taken from the mesenteric glands, kidney, spleen and heart-blood, indicating that *B. coli* had not only invaded the small intestine but the body as well.

Microscopic sections stained with Giemsa showed enormous numbers of bacteria resembling *B. coli*, extensive invasion and necrosis of the mucosa, with hemorrhagic edema and gas bubbles in the submucosa.

From the bacteriological and histological studies, it appeared

that the disease was the result of invasion by *B. coli*, which had assumed pathogenic and invasive properties.

Agglutination tests with Brucella antigen indicated the widespread prevalence of brucellosis in the herd and it is believed that this disease was a primary factor responsible for the birth of many pigs below par, with a lowered resistance, rendering them liable to infection by *B. coli*.

In the absence of any definite or well-supervised system of breeding, it is believed that prolonged inbreeding also may have been a factor responsible for the farrowing of weak pigs of low vitality.

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[2]Smith, T., and Orcutt, M. L.: The bacteriology of the intestinal tract of young calves with special reference to the early diarrhea ("scours"). Jour. Exp. Med., xli (1925), 1.

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[4]Hayes, F. M., and Traum, J.: Preliminary investigations of three outbreaks of swine abortion in California. No. Amer. Vet., 1 (1920), 2.

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[6]Howarth, J. A., and Hayes, F. M.: Brucelliasis in the swine herd of the University of California. Jour. A. V. M. A., lxxviii (1931), n. s. 31 (6), pp. 830-848.

Discussion

Dr. Ward Giltner: As usual, this paper by Dr. McBryde is very excellent. I wish we might have some information eventually, if you have saved any of these cultures, on exactly what colon organism it is, whether it is paracolon or just exactly what it is, because it will be of importance to those who follow this up.

I want to call the attention of those here to the fact that with the plate method, which Dr. McBryde used, if you get a good antigen, nothing could be simpler than the diagnosis of this Brucella disease in swine. It can be done very rapidly and with a fair degree of accuracy, with whole blood.

Dr. McBryde: I used blood serum. The blood was allowed to coagulate and was left overnight in the ice-box.

Dr. Giltner: If you can not resort to that, you can make a very good diagnosis with whole blood and it is possible to do it in the slaughter-house.

I might say that this is the first instance that I have heard recorded where this complication, coli bacillosis, has entered into the Brucella disease.

Dr. I. E. Newsom: I would like to ask Dr. McBryde if any suspicion was cast on the nutrition—that fact that these were garbage-fed hogs? We have had a similar condition in garbage-fed hogs around Denver for a good many years, and in trying to work it out we had considerable correspondence with the veterinarian who formerly was in charge of this ranch of which you speak. In those days it was felt that, while colon bacilli could be isolated readily, they were at least not the primary cause. The way the garbage-feeders around Denver have worked it out is to put these sows on feeds other than garbage for practically a month before farrowing. I was under the

impression at that time that that was the system in use on this California ranch. The manager said that it was routine procedure for them to take these sows at a certain period before farrowing and put them on feed other than garbage and he felt that they could more nearly control the problem by doing that; in other words, by controlling the nutrition, than by attention to sanitation or any biological methods. That was the way in which the problem finally was worked out among the garbage-feeders at Denver, and those men still find that, if sows are fed on garbage up to the time they farrow, the pigs develop enteritis on a much larger scale than if changed to grain feed.

DR. McBRYDE: The ranch veterinarian had carried out some very extensive experiments along nutritional lines before my arrival and felt satisfied he could not eliminate the trouble by feeding different rations. He took the sows off garbage entirely for several weeks before farrowing and got no results whatever. I was informed that a veterinarian, formerly in charge of the veterinary work at the ranch, had stated that the disease existed on the ranch when he was there, and he thought that it could be eliminated by a change in the diet—by simply cutting down the diet of the sow, not taking her off garbage, but reducing the diet prior to farrowing. The present ranch veterinarian, however, tried that without any success at all. He had worked pretty thoroughly on sanitation and nutrition and had done a great deal of very good experimental work along these lines, but without achieving any results.

DR. B. S. FRITZ: I would like to ask Dr. McBryde if the strain of the abortion organism was identified? Was it the strain we are dealing with, and what percentage of losses were experienced from the abortion organism?

DR. McBRYDE: I did not isolate the abortion organism. It was almost impossible to obtain premature pigs or placentae from which to make cultures, as the heavy sows were kept in large pastures until within a few days of farrowing. The broodermen said the sows ate the little pigs and afterbirths very soon after they aborted.

I do not believe they had so many abortions, but it was very hard to check that. Either the broodermen did not watch the pastures, or the sows ate the pigs. I could not get any definite data on that.

DR. H. J. SHORE: I would like to ask Dr. McBryde the cause of the general belief that this abortion strain may be pathogenic to humans. Are there any cases of undulant fever among people around there?

DR. McBRYDE: One of the broodermen had been in poor health for a year or so, but I do not think his blood had ever been tested. I do not know of any definitely positive cases, but I rather expect there may have been some that passed unrecognized.

Veterinarian's Son an Artist

The drawing that features the holiday announcement of Ashe Lockhart, Inc., in this issue of the JOURNAL, is the handiwork of Philip Stone, son of Dr. W. J. Stone, prominent veterinarian of Joplin, Mo.

New York—August 13-18, 1934
12th International Veterinary Congress

COMMON AILMENTS OF SHEEP*

By F. E. STILES, *Battle Creek, Mich.*

When Dr. Hershey, our Secretary, asked me to take part in this program, I hesitated, not because I did not want to do my part but because I felt that someone else would be able to bring you something more scientific and up to date. My remarks will cover only ailments with which I have come in contact during my practice. A few general remarks about sheep at this time might be profitable.

Just treating the individual sheep or lamb occupies a very small place in handling sheep troubles. Further, they are about the most sensitive animals that a veterinarian has to treat and the most susceptible to shock. They are dainty as to diet and have a keen sense of smell. The normal sheep has a pulse rate of from 70 to 90 and a temperature of 103° F. A little excitement can change this pulse-rate. So bear in mind always to approach a flock of sheep quietly and, in handling them, never to grab them by their wool. These are little things, but often attention to details and the art of doing a job well have considerable to do with the prestige we gain with the stock-owner.

For the sake of system and brevity, I will divide my paper into three parts: ailments of the breeding ewe; ailments of baby lambs; and the feeding of lambs.

NODULAR DISEASE

Nodular disease seldom is recognized until the ewe or ewes are beyond help. This disease is caused by the *Oesophagostomum columbianum* and is on the increase.

Symptoms: General debility, emaciation, anemia, and weakness to the point where they often lie around, still eating and growing thinner until death ensues. This disease is chronic and will persist in a flock a number of years, causing severe losses each year until an ordinary owner gets discouraged and quits sheep-raising. Postmortem findings are familiar to you all. The nodule in the wall of the intestine is caused by the burrowing of the parasite during a certain stage of the life cycle. Several years ago, these nodules were considered a sort of adenitis. In the course of the disease, the nodules become so numerous that the absorption of nutrition from the intestine is well nigh impossible.

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*Presented at the seventieth annual meeting of the American Veterinary Medical Association, Chicago, Ill., August 14-18, 1933.

Treatment: Separate the affected animals from the rest of the flock, as they cannot stand crowding and pushing about. Give them a diet especially nourishing. Arsenic and iron tonics help some. If they can be pulled through the winter, it is well to change the grazing grounds in the spring. I have observed that flocks regularly drenched for stomach worms rarely are badly affected with nodular disease.

Stercoremia

This disease always affects the pregnant ewe, usually one carrying twins. The patient in most cases is a thrifty, fat and healthy-appearing animal until the disease—which is sudden in onset—takes hold.

Symptoms: A careful observer will notice dullness for perhaps one or two days before the active symptoms appear. These are: the grating of the teeth, champing of the jaws, more or less salivation and an unsteady gait. The stricken animal soon is down, paralyzed, and lies this way from three to five days.

Treatment: Treatment of the affected animal is not practical, as it usually is unsuccessful.

Cause: Lack of exercise, combined with plenty of food, and pregnancy. The patient apparently gets bilious and certain toxins are absorbed from the intestinal tract, causing the nervous derangement. Postmortems show nothing except the possibility of a slightly solid condition of the fecal matter in the cecum.

Treatment of flocks unaffected but susceptible: If the ewes have been kept in a small yard or shed, get them out at once and see that they get plenty of exercise. Use your ingenuity in obtaining this. I like to have the owner take them from 60 to 80 rods from the building and scatter their feed out. In my state—Michigan—snow is usually with us at the time this disease appears; so we scatter the hay or fodder on the snow. Under certain conditions, driving may be necessary. I see where one investigator advises ten miles a day, but I feel that is rather the extreme. Also, I make it a practice to give each sheep one ounce of Epsom salt mixed with common salt, on the grain, once daily for three days. This simple treatment is very successful and always will stop progress of the disease in the flock.

Foot-Rot

This condition is well known to every practitioner who treats cattle or sheep. I am sure we have two distinct types. One responds readily to antiseptic and astringent treatment. This type probably is caused by pus-producing groups of organisms. The

other type is due to *Bacillus necrophorus*, and will tax the ability and ingenuity of the veterinarian if recovery is to be brought about. I might add that this affects the feeding lambs as well as the ewes, and, unless looked after early, causes severe loss in weight in feeders. The death loss, if any kind of treatment is given, is practically nil.

Treatment: In small flocks the treatment may consist of cleaning up the feet and applying a good antiseptic astringent. Usually I use 10 per cent copper sulfate containing about ½ to 1 per cent of formalin. This is applied with a cotton swab. In a large flock, this treatment is impractical. In this case, the best method is to drive the sheep through troughs containing medicaments.

Rabies

This disease, while not common, may confront the practitioner. I had an interesting experience with this disease in a flock of ewes last winter. The owner called me on Christmas day, saying that some of his sheep had gone crazy. One of the ewes had butted his father and had hurt him quite badly, and several others were on a rampage. The history revealed that a stray dog had passed through the flock three or four weeks before and had bitten several of the sheep.

Symptoms: An interesting symptom of the disease was the sexual excitement exhibited in several of the ewes. They were jumping other members of the flock like very amorous rams. We separated the affected animals from the rest of the flock and put them in a small shed. They started butting the walls of the shed and kept it up for two or three hours, until they fell down in a paralyzed condition and died within twelve hours.

Diarrhea of Baby Lambs

This disease is usually fatal. It is caused by insanitary conditions and most frequently appears during damp spring weather, when the bedding gets wet and the udders of the ewes become soiled. To prevent the disease, neat, clean, airy quarters should be provided and the udders of the ewes disinfected.

Stiff Lambs, or Pseudotetanus

If the lambs have been docked recently, this disease at first is quite apt to be diagnosed as tetanus. A careful examination reveals some differentiating clinical symptoms, however. For instance, in tetanus the eyeball is covered with the nictitans membrane, a condition not seen in stiff lambs. Furthermore, in a

stiff lamb, percussion does not evoke spasms as is the case in tetanus. Some affected animals just show a stiff gait, similar to that shown in recently castrated lambs; others go down and lie paralyzed.

The history of the case is very important. This condition shows up three to ten days after the lambs have been placed on pasture. It is not observed in lambs born out in the pasture. Fifty per cent of the lambs that go down with the disease will die; the others may make a spontaneous recovery. I had several cases last spring. I tried calcium gluconate but without results.

The Feeding of Lambs

The feeding of lambs is one matter; the handling of them is quite a different picture, and I could easily spend my allotted time discussing the different phases of that important operation —fattening lambs for market.

First, I would mention the numbers handled by our feeders: one to ten cars, giving us 300 to 3,000 animals. Some men are always going into the sheep-raising business because a neighbor, a season or two previous, made a lot of money. More often than not, they sustain terrific losses.

I would state that 95 per cent of the losses are due to dietary disturbances, notwithstanding the fact that we have shipping fever, quite similar to that of feeder cattle, called by various names, an old favorite of a few years ago being hemorrhagic septicemia. In certain bands, the veterinarian must contend with stomach worms and tapeworms, scab, keratitis and foot-rot, but, in spite of all this, I still hold that the major losses are traceable to dietary disturbances.

The principle of diet is the same, although it varies on the different farms. In most cases, too much heavy grain is consumed, along with insufficient succulent roughage. If feeding lambs have access to *plenty* of good quality alfalfa or clover hay, they will not engorge themselves on grain nearly so often. It may be that some of you have heard the statement that lambs could not be put safely on shelled corn self-feed, but I must contradict that. With the proper set-up it can be done. First, the lambs must weigh 60 pounds or more; then, good hay fed in the self-feeder to the extent that 20 per cent is wasted. You will get along pretty well, but be mighty careful that good hay is before them at all times.

Cornfield lamb-feeding trouble begins from 14 to 17 days after they are turned in the field.

Shipping Fever

Shipping fever comes on from three to ten days after the animals have arrived from the public stock yards. The symptoms are similar to those in cattle. If the sheep are carefully handled, the losses are not heavy.

Internal Parasites

Early diagnosis and careful, systematic drenching usually will control internal parasites effectually.

Scab

In scab, early diagnosis and dipping are necessary. I would like to make a point here about finding mites. A cold, wet day, if the patients have been exposed, makes it difficult to locate the mites, which normally are found around the edge of the lesion.

Keratitis

Symptoms: Acute inflammation of the cornea of the eye. The onset is sudden, and the disease spreads rapidly if the sheep are crowded together, as in feeding sheds. The acute inflammation of the cornea is accompanied by lacrimation and a cloudy appearance of the eye. Quite a few of those affected go blind temporarily.

Treatment: In those recently affected bathe the eyes with a solution of boric acid and then instill a few drops of a 20 per cent solution of zinc sulfate. This is a specific in the early stages of the disease. Cauterize corneal ulcers and follow with daily application of a 2 per cent yellow oxide of mercury ointment and gentle massage. Thoroughly disinfect the feed-rack and drinking-trough. The results of this treatment are satisfactory if it is carried out thoroughly. Here, again, the fatalities are few, but losses in weight often are severe.

Lip-and-Leg Disease

This disease occurs frequently in feeding lambs and, to the uninitiated, might cause quite a little anxiety, as it might be confused with foot-and-mouth disease. I have had some clients quite excited about this condition.

Symptoms: A sore on the lips, covered with a crusty scab. The underlying tissue bleeds easily after the scab is removed.

Treatment: Since this disease is caused by an anaerobic organism, the most important phase of the treatment is to remove the scab. This is done best if the operator wears rubber gloves and uses a corncob. After removing the scabs, apply a disin-

fectant such as some of the coal-tar preparations. If treatment is applied early and thoroughly, usually one is sufficient. If the cases are allowed to progress to the point where both upper and lower lips are involved, as well as portions of the face, considerable patience is required to effect a recovery. It will be necessary to wash the involved spots daily and apply a mild ointment, such as zinc oxide. This is a disease that usually is not fatal, but if it is allowed to take its course in a band of fattening lambs it will cause a marked delay in putting on weight. A further precaution is to separate those affected with the disease from the healthy. Disinfect the feed-racks and the trough, although this procedure is not so essential as in some other diseases.

Laminitis

This disease is caused by overeating, on a concentrated diet.

Symptoms: Lambs usually are observed down on their knees, crawling about. Many of them maintain a pretty fair appetite and usually recover without any treatment.

Alopecia

Alopecia (loss of wool) is really a symptom and not a disease. It is the result of some other severely debilitating ailment. Then, when the patient begins to thrive, he loses his fleece. No treatment is required except protection from storms or cold.

Obstetric Hints

First, a case of dystokia can be handled much more easily if the patient is put on a table. This usually is an improvised affair but you will find it much more satisfactory to work on the patient on a table about three feet high than on the ground. You can do better work and carry out your manipulations better. The ovine obstetrician requires very few instruments, as most all of the work is done with the hands alone. A pair of good forceps will be used occasionally.

In closing, the point I wish to stress is the susceptibility of the ewes to shock. This is the tip to the young practitioner: Always warn your client of the possible outcome. Many times, I have handled a difficult dystokia case in an apparently successful manner, only to have the patient expire shortly after the operation was completed.

Discussion

Dr. F. M. Nelson: We have had a lot of experience with foot-rot out in Montana, and we find that copper sulfate, mixed as strong as you can mix it in boiling water, is not too strong. The main part of your treatment in foot-rot is trimming the foot. You must trim away every

bit of diseased tissue. If the whole hoof drops off, that sheep will get well more quickly than when you have to cut.

Personally, I handled something like 50,000 head of sheep last winter. Many of these sheep were affected with foot-rot and we found in that work that you have got to get every bit of the loose hoof trimmed off. That foot should be soaked for not less than two minutes in a saturated solution of copper sulfate in either water or vinegar, and you will get recoveries in a large percentage of the cases providing you move them to clean ground. They have to get off that infected ground; the foot has got to be trimmed properly—and don't be saving with your bluestone. We have a lot of it left in the bottom of our crocks or barrels all the time, and it is surprising how soon that sheep will walk on that foot if it is properly treated.

DR. J. C. FITZPATRICK: I noticed you mentioned the feeding of alfalfa and clover. What about the feeding of soy beans?

DR. STILES: I really haven't had much experience in feeding soy-bean hay. Some of our feeders planted soy beans in their fields along with corn, and it has worked out very nicely.

DR. FITZPATRICK: We had quite a bit of trouble with some western feeders last year that were being fed on soy-bean hay. I had an experience similar to what you said about the overfeeding of grain, and I was wondering whether or not they would get as much of the soy bean for roughage as they would of alfalfa hay. We had these sheep almost entirely on soy bean hay, and we had quite a bit of trouble with them.

A Wonderful Discovery

Sometimes we get a bit impatient at the progress, or lack of it, being made in the control of some of the more common animal diseases. However, when a discovery is made that promises relief from several diseases, animal and human, all at the same time, then we sit up and take notice.

One of our members connected with a large educational institution recently received a communication that offers encouragement to dairymen, even though there is a big surplus of dairy products at the present time. Here it is:

> I am dropping you a line to say that I have a cure for contagious aboition. I can take all germs from milk such as, tuberculars, typhoids, scarlet-fever, after five day treatment it will stand a test of any chemist in the world. It will (the cure) make cows give more milk and cream.
>
> I also have found many different elements in milk that know one else knows about. These articles which I have referred to, I would like very much to demonstrate. I can assure you that once the cows are treated the germs will never come back. It is very hard to write and tell you about everything I have discovered, due to the fact it has never been published in books and no name given to them.
>
> I want to say that this disease I have followed for (55) fifty-five years, I am now (74) seventy-four. I have followed it in hogs, cows, sheep and horses. I will close hoping you will take this letter into consideration.

We promised not to publish the name and address of this 74-year-old investigator until we could look into the matter.

GAS GANGRENE AND TWO CASE REPORTS IN CATTLE*

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Gas gangrene includes a number of wound infections characterized by edema with emphysema, often terminating in gangrene and sepsis. Anaerobes play the principal, if not the only, rôle in its production. It is known also as gas edema and gas bacillus infection. Two forms in animals are familiar, *viz.*, blackleg and malignant edema. Gangrene is not always present in gas edema.

The disease was described first by Maisonneuve, in 1853. Battin demonstrated its infectious nature in 1871. One of the causes of the disease was discovered by Pasteur, in 1873, and named by him *Vibrion septique*, which is now considered synonymous with *B. edematis maligni* of Koch. Welch[1] was one of the first American investigators to study it. In 1891, he discovered and named *Bacillus aerogenes capsulatus* as the causative factor. This organism later was named *Clostridium welchi*, a name which it still holds.

Welch found that it was an anaerobe and could be obtained from infected humans from:

1. Gas bubbles in the blood and organs (after death).
2. Gaseous abscesses.
3. Emphysematous gangrene.
4. Such uterine infections as:
   Emphysema of the fetus.
   Puerperal endometritis.
   Physometra.
   Emphysema of the uterine wall.
   Puerperal gas sepsis.
5. Various infections of the urinary tract.
6. Infections derived from the intestinal tract as:
   Local gastrointestinal lesions.
   Pneumo-peritonitis with and without perforations.
   Hepatic and biliary infections, intestinal emphysema of the gastrointestinal, genito-urinary and biliary tracts.
7. Pulmonary and pleural infections.
8. Blood during life.
9. Meningitis.
10. Cavities in the brain.

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No such exhaustive research has been reported in the various species of animals as that reported by Welch. We are more or less familiar with some of the symptoms and lesions mentioned by him. It has been the custom in veterinary medicine to diagnose such conditions as malignant edema. Malignant edema and blackleg now are considered specific forms of gas gangrene. It was not known until recently that sheep were susceptible to blackleg.

Marsh has done some excellent work on gas gangrene in sheep and has published his findings.[2-4] He identified the cause of the disease as *Clostridium chauvoei.* Bacteriologically and pathologically he found the disease to be identical with blackleg in cattle, the only difference being that in sheep it seldom if ever occurs spontaneously as it does in young cattle. He states, however, that gas gangrene or blackleg in sheep may be due to other gas-producing organisms and that when an animal receives a wound of a nature favorable to the development of anaerobic bacteria it is more or less a matter of chance whether *C. chauvoei, V. septique* or some other anaerobe will be the invader.

Roberts and McEwen,[5] of England, have studied gas gangrene in sheep and have confirmed the findings of Marsh.

Weinberg and Seguin[6] made a comprehensive report of their studies of gas gangrene in wounded soldiers during the World War. They found that the condition was due to one or more species of anaerobes and that the most common ones were identified as: *B. perfringens, B. sporogenes,* and two new species described under the names *B. edematiens* and *B. fallax.*

The Medical Research Committee,[7] in its report upon anaerobic bacteria and infections, stated that the principal pathogenic species of anaerobes are: *B. welchi, V. septique* and *B. edematiens.*

The symptoms of so-called malignant edema are easily recognized and much dreaded by men in practice.[8,9] The disease is well described in veterinary textbooks.[10,11] However, it is the opinion at present that one is not able to diagnose malignant edema from symptoms and lesions alone, but a bacteriological examination is required to establish an exact diagnosis. Kelser[11] seems to think that some of the malignant edema-like diseases really are not malignant edema, but are caused by *B. welchi,* an organism which is distributed widely in the soil.

Gas gangrene belongs to that group of diseases designated as soil diseases. The various anaerobes that cause this condition are found quite widely distributed in the soil and when they find

entrance into the tissues may set up a disturbance. This is particularly likely to be the case when conditions make anaerobic growth possible. Once the anaerobes become implanted, the tissues are invaded by a new growth of organisms with the formation of toxins that damage tissue and facilitate further growth and damage.

When malignant edema organisms find suitable conditions in the tissues, they multiply rapidly and usually terminate promptly in sepsis and death. They are indigenous to the soil and in feces and often are present in cadavers a few hours after death in animals that have died from various causes. In order to prove that death was due to the bacillus of malignant edema, specimens should be procured before or soon after death.

It is impractical for one in general practice to give sufficient time and attention to each case of suspected gas gangrene to determine its true etiology. Most cases are easy to recognize from the history, symptoms and macroscopic findings, but the assistance of an experienced bacteriologist is required to determine the kind of organism responsible for the disease.

Some investigators report that malignant edema is rare in cattle, yet we know that the symptoms of it are not rare. It probably would be best to make a tentative diagnosis of gas edema, which is a broader term, and wait for the autopsy and the laboratory to establish an exact diagnosis.

Horses, cattle, sheep and swine are susceptible to gas gangrene following certain injuries due to careless or unskilled surgical technic in castrations, docking, inserting setons, vaccination, dystokias, lacerations from sheep-shears, etc. Infection occurs much as it does in tetanus. Gas gangrene and tetanus may develop from the same injury.

Among the first symptoms to appear in gas gangrene are a crepitating swelling, elevated temperature and depression. There is no fever in an ordinary edema and no crepitation in the usual forms of inflammatory edema. In most forms of gas gangrene septicemia appears a few hours before death.

The disease is much less common than in former times or before aseptic and antiseptic surgery were understood. While gas gangrene seldom is diagnosed in practice, it is believed to be more common than generally is suspected. The cause of death in selected cases, if submitted to laboratory examination, might prove to be due to some of the numerous forms of anaerobes that cause the disease.

Ways in which infection occurs are known. The symptoms and lesions are not difficult to recognize. The treatment is not so

easy, especially after septicemia has developed. Swellings should be opened freely to admit all the air possible to the affected tissues. Biological preparations combined with a suitable surgical technic were used with success in treating infected soldiers in the World War. With the prompt use of biologics, it was believed that the disease could be prevented. Some benefit was reported from their use even after the disease had developed.

With the exception of blackleg, vaccines have been used but little as preventive or curative agents in any of the forms of gas gangrene found in animals. We hope, in time, however, that vaccines will be developed for the other forms of infections.

Case Reports

During the spring of 1933, two high-producing, pure-bred, dairy cows were brought to the Veterinary Hospital as patients. Gas gangrene was diagnosed in the first patient, and a tympanites, possibly followed by gas gangrene, in the other.

Case 1: This was in a pure-bred Guernsey, freshening normally on February 1. She came down with parturient paresis the following day. The udder was inflated promptly but the patient failed to respond completely. Calcium gluconate was given intravenously with but slightly better results. Both treatments were repeated on the second and third days. The animal could get up with assistance, but was decidedly lame in the right hind. There was considerable emphysema in the subcutaneous connective tissue of the udder and a crepitating swelling was observed extending from the hock to above the region of the patella. This condition developed soon after the udder inflation and the attending veterinarian was apprehensive that he may have forced too much air into the udder and ruptured some of the acini and that a part of the air had escaped into the subcutaneous tissue. Emphysematous swellings were observed later over the loins and other parts of the body.

The appetite was fair, but the animal became more jaded during the following two weeks. She was brought to the hospital in a truck on February 15. Upon arrival, she was prone and unable to get up even with assistance. The temperature was 102.6, pulse 104 and the respirations 68. Samples were drawn from one of the emphysematous areas for laboratory examination. Macroscopically the sample was a dark brownish blood serum filled with small air bubbles. From the physical symptoms and the laboratory findings a diagnosis of gas gangrene was made.

During the following five days, the temperature ranged from 102 to 104, the pulse from 76 to 112 and the respirations from 20 to 76.

On account of the pronounced lameness or inability to use the right hind leg, the extensive swelling and an occasional deep-seated crepitation in the region of the patella, a fracture was suspected. The x-ray failed to show any and none was revealed at autopsy. The skiagraph, however, showed considerable gas and fluid in the part. It was believed that the crepitation was due to tendovaginitis sicca.

Treatment: It was too late for treatment when the animal arrived at the hospital. No encouragement for a recovery was given. The swellings were opened freely, stimulants and anti-gas gangrene serum were administered. The swellings subsided but the symptoms of septicemia became more pronounced. The animal became moribund and was destroyed on February 22. An autopsy followed immediately.

Autopsy findings: The right hind leg showed an opening (surgical) on the inner side of the hock joint from which a foul-smelling fluid exuded. The tissues at this opening were soft and brown in color. There was a slight crepitation in a swelling on the right front leg and much crepitation in the right hind.

The hind leg was removed and upon examination it showed fistulous tracts between the various muscles extending up to the great tuberosity of the femur and contained a dirty brown, foul-smelling material. The individual muscles were a dirty brown on their surface and on cut sections had a cooked appearance.

The articular cartilage at the head of the tibia showed some softening and the periosteum was gray in appearance. There was necrosis of the periosteum on the inner side of the tibia and of the articular cartilage on the proximal end of the tibia. The anterior tibial artery was occluded completely with clotted material and its walls were swollen and soft.

The intestinal tract showed catarrhal enteritis. The liver and kidneys were swollen, pale in color, soft and friable, indicating degeneration. The lungs showed great accumulations of air in the interstitial tissue and air-sacs, indicating an interstitial and alveolar emphysema.

The autopsy confirmed the diagnosis of gas gangrene. Numerous organisms were found but these were not identified.

Case 2: This was a three-year-old Ayrshire. She calved normally on March 14 and milked well until April 2, when she was slightly off her feed. A pronounced case of tympanites developed

the next day. The local veterinarian was called and found tympanites of the rumen and also of the pharyngeal region. The gas was removed from the rumen with a trocar and canula about 11 o'clock. At 2:30, the ambulatory clinic saw the case in consultation with the attending veterinarian. The rumen and parotid region were fully distended with gas at that time. The rumen was tapped and an unsuccessful attempt was made to pass a probang. It met an obstruction in the esophagus, about six inches posterior to the pharynx. On palpation of the pharynx and the anterior portion of the esophagus, several handfuls of food were removed. No foreign body was found.

The attending veterinarian gave as his opinion that there was a rupture of the esophagus and that the gas in the parotid region was escaping from the rumen through the rupture. The emphysema at this time extended nearly the whole length of the jugular groove and well up the sides of the neck.

The patient was taken by truck to the Veterinary Hospital, arriving there about 5 o'clock in the afternoon. An x-ray failed to show any obstruction in the esophagus, but showed considerable gas in the region. A second unsuccessful attempt was made to pass the probang. About two hours later, it was passed easily and a sufficient distance to reach the rumen, but no gas or stomach contents came through it.

The next morning there was practically no gas in the rumen. Peristalsis was inactive, temperature 100.8 and pulse 120. Food and water were refused. A rumenotomy was performed about 10 o'clock, April 4. Nothing abnormal was found. The patient died 24 hours later. The autopsy followed promptly.

Autopsy findings: An extensive emphysema and edema appeared in the connective tissue of the head and neck. All four stomachs were distended with gas. There was a brownish serum containing clots of blood in the thoracic cavity. Considerable gas was found around the kidneys.

A gelatin-like material and food particles were present in the connective tissue between the esophagus and trachea anterior to the bifurcation of the trachea. The esophagus was ruptured dorsally and ventrally. Each wound was clean cut, two inches in length, and located about eight inches posterior to the pharynx. There were two other wounds in the esophagus, each about one and a half inches in length, extending through the mucous and muscular coats, one just posterior to the pharynx and the other about four inches lower. All of the four wounds were parallel and longitudinal with the esophagus. They had the appearance

of having been cut with a sharp instrument. Nothing was found in the surrounding tissue or in the contents of the digestive tract that could have caused these wounds.

The mediastinal tissue was edematous and necrotic and filled with a reddish-brown, gelatinous material. The mediastinal lymph-glands were swollen and edematous. The parietal pleura was covered with reddened areas and pseudomembranes, with an acute serofibrinous pleuritis. There was a beginning gangrene of the inner surface of the lungs.

The right upper border of the liver showed a pale yellowish color and evidence of degeneration. The cortex of the kidney and the tubules stood out prominently on the cut surface.

A small perforation of the reticulum by a wire was found. The heart showed hydropericardium, acute pericarditis and emphysema of the pericardial sac. There were hyperemia and granulations on the epicardium and an extensive subendocardial hemorrhage.

From one of the ruptures in the esophagus there was a tract which extended along the side of the esophagus for about 18 inches and contained particles of food.

It was our opinion that the esophagus was ruptured as a result of an obstruction or a stricture before the local veterinarian was called, yet there was no history available to this effect. The probang eventually entered one of these openings and passed easily through the loose connective tissue between the trachea and esophagus and to a point near the diaphragm.

It is probable that this case started with choke, followed by tympanites and rupture of the esophagus. Conditions were favorable for anaerobic infection. Some of the symptoms and lesions would indicate that gas gangrene had developed before death. Specimens were not submitted to the laboratory for examination. For this reason the diagnosis remains problematical.

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POULTRY AND THE PRACTITIONER*

By NORMAN W. ACKERMAN, *Van Horne, Iowa*

Since the time that veterinary medicine became known as one of the arts or sciences, we who have been engaged in that work have been constantly forced to revise our whole conception of the field of medicine. New discoveries in regard to the nature and cause of disease and new methods of treatment, with constantly changing economic conditions, have changed our sphere of activity until we are engaged in a varied line of work that would cause a practitioner of thirty years ago, if suddenly transposed to our present day, to wonder if he were a member of the same profession. In the field of poultry production this is especially true, and we can well remember, but a few years ago, when many of our most capable practitioners openly made light of any veterinarian who even attempted to do any poultry work. Today the pendulum has swung until any general practitioner who fails to avail himself of this phase of our work finds himself seriously handicapped, for the poultry industry has grown by leaps and bounds and its members are insistent upon proper guidance in their work.

In considering the relationship of poultry and the practitioner, we shall have several comments to make that seem pertinent to us. It is our hope that they will be received in the spirit in which they are given—that of constructive criticism.

The great need of the veterinary profession today is that the practitioner learn something about poultry husbandry. As a group our knowledge of the normal fowl and its care is pitiful. Nor is the practitioner entirely to blame, for our schools have been very slow to take up this phase of the work and many of them neglect it entirely, except in a purely cursory way. If they fail to prepare their graduates properly, is it any wonder that the student fails to do his school credit in this particular portion of our work? In no phase of our activities is the practitioner called upon more frequently for advice than regarding the care, feeding and raising of poultry, and in many cases he is the means of no inconsiderable financial gain or loss.

One of our members, who annually meets a large number of practitioners in a personal way, made the statement not long ago that barely one of ten veterinarians whom he knew was capable of giving anything approaching good advice to a client,

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because they did not know what to do themselves. If this is the case, and we have no reason to doubt that it is so, is it any wonder that as yet we have failed, generally speaking, to gain the coöperation of those engaged in poultry husbandry? We frequently hear someone loudly bewailing the fact that this or that agency is encroaching on our domain. The truth is that in the past we have been tried and found lacking and clients were forced to turn to other sources of information. When we, as a profession, can give our clients the advice they require, then, and then only, can we hope to compete with outside influences and encroachments. In this case it is going to be doubly hard, due to our own past neglect and indifference.

The great distances and climatic changes of our country, as well as the varied phases of poultry production, have brought about a great number of problems, some of which are of high importance in some districts and negligible in others. Generally speaking, no hard and fast rule of normal production may be made, for that which may be beneficial in our southern areas would not be the proper method in the north and eastern sections. Therefore, to a great degree, each section of our country must work out its own method of normal production, the only rule being —Is it satisfactory?

Much Valuable Research Work

As a profession we need have no apologies to offer for either the quantity or quality of the research work being done to combat the diseases affecting the domestic and wild fowl of this country. In our section of the Middle West, the diseases met with are relatively few. These include such conditions as fowl cholera, typhoid or pullorum disease, roup, fowl-pox, tuberculosis, coccidiosis and parasites and will cover a large majority of the cases presented to the practitioner for diagnosis. In late years infectious leukemia and infectious laryngotracheitis also have been assuming more prominent positions. The practitioner can find splendid articles on these in our text-books and journals, and failure to have a good working knowledge simply means a lack of interest on our part. It is our belief that, a great share of the time, trouble in poultry is caused more by mismanagement than disease and we must be in a position to know whether this is the case or not, else we lay ourselves open to grave error in treatment.

It also has seemed that, in the past, there has been a great tendency towards discovering the etiology and describing the pathology of a condition and letting the treatment take care of

itself or leaving it to the ingenuity of the practitioner, or to some of our supply houses to devise some mode of treatment, instead of following through to a logical conclusion in the form of an effective and applicable method of treatment. It is very true that for many of our conditions we have biologics more or less efficient, but the poultry-raiser will use them only as a last resort, preferring to spend his money for drugs and nostrums so widely used and advertised. The practitioner must either comply with the wishes of his clients, or find them patronizing the drug store or "specialist," who travels about from farm to farm. It is true also that our biologists do not always do what we expect of them and it is hard to convince a client, after that happens, that the correct procedure has been followed, there being many who will be eager to foster that idea in their minds. They also have something to sell besides their services.

Many Unsolved Problems

In the field of infectious diseases there remains much to be done. For instance, consider the case of fowl-pox; some investigators make the assertion that fowl-pox and roup are of the same origin or rather have the same causative factor. If so, why do we find so many outbreaks of roup with no lesions that can properly be classed as those of fowl-pox and outbreaks of fowl-pox in which we find few if any lesions of roup, or avian diphtheria, as it is more properly called? We have had some splendid work done showing that mosquitoes are capable of transmitting this disease and yet we do not remember ever having read or heard of the importance of other birds as carriers, such as the common English sparrow; or the fighting among birds, especially the lighter breeds, in which the disease seems to spread more rapidly, nor to the act of copulation, during which the male often grasps the comb of the female causing an abrasion of the skin and opening an avenue for infection.

Many years ago, when the study of poultry diseases was in its formative stage and attempts were made to classify the various diseases of poultry, there was one that caused more trouble and loss than any known then or now. One of its predominant characteristics was that of diarrhea and it became known as bacillary white diarrhea. It is now called pullorum disease. There was also a disease in the mature fowl, of bacterial origin, the organism in every way resembling that causing pullorum disease in the young chick. This has been called avian typhoid. Many have felt, in late years, that the two are one and the same disease. Personally we feel that they are and, if so, why con-

tinue to separate them and add to the confusion, simply because the earlier investigators made a separate classification?

So, through the field, we find many instances where work should be done both by and for the practitioner, that the situation may be clarified and we be enabled to be of greater value to the industry we serve.

12th International Veterinary Congress
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West Virginia Eradicates Bovine Tuberculosis

West Virginia is now in the modified accredited area, signifying its practical freedom from bovine tuberculosis. This designation by the U. S. Department of Agriculture became effective December 1, 1933, promptly following the successful completion of testing of all cattle in Roane and Wirt counties. All other counties had qualified previously as modified accredited areas. West Virginia thus became the thirteenth state to eradicate tuberculosis.

Tuberculosis-eradication work was taken up in a limited way in West Virginia in 1919 and more actively about 1923. Good progress was made in recent years in spite of the difficulty of traveling over some of the mountainous parts of the State, and the fact that the cattle were widely scattered. The degree of tuberculosis infection was slight in most of the 55 counties of West Virginia.

NEW VETERINARY HOSPITAL AND CLINIC BUILDING AT TEXAS A. AND M. COLLEGE

GASTROTOMY, ENTEROTOMY AND CYSTOTOMY, AND THEIR INDICATIONS IN THE DOG AND CAT*

By CHAS. W. BOWER, *Topeka, Kan.*

This is a subject that deals with operations of great importance to the small-animal practitioner. While we all remember discussing these in the classroom and studying them from our surgery text-books, yet we were handicapped, inasmuch as there was very little information as to their application to the dog and cat.

It is not the writer's intent to give an elaborate discourse concerning these operations, neither is it his intention to offer any new technic, but instead he wishes to stress the importance of having this knowledge at hand so that it may be used at a moment's notice. The writer furthermore would like to have it clearly understood that these operations will be discussed purely from a practitioner's viewpoint.

Before entering into the technic of and indications for these operations, let us have a definite understanding as to their real meaning: Gastrotomy means incision into the stomach; enterotomy, likewise, an incision into the intestines; and cystotomy, incision into the urinary bladder. In general, the indications for opening these organs are for the purpose of removing something. It may be a foreign substance or a piece of necrotic tissue. However, the indications will be discussed more specifically in a later paragraph.

The technic of any operation varies with the operator. However, it will be the writer's endeavor to outline as simple a method as possible.

GASTROTOMY

The patient is placed into profound sleep by use of a general anesthetic. Whether an injection anesthetic or an inhaling anesthetic is used makes very little difference; personally the latter is preferred. The landmark for the laparotomy incision is the space between the cartilage of the sternum and umbilicus. This area is shaved and cleaned with ether and alcohol and finally painted with tincture of metaphen or iodin. A sterile sheet should be placed over the animal, with an opening over the area just prepared. The incision through the skin and abdominal wall is made on the median line, about two to three inches posterior to the cartilage of the sternum and extending backwards

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almost to the umbilicus. The length of this incision may vary with the size of the patient.

The muscle and peritoneum are picked up by small artery forceps and held outwardly by an assistant. The stomach is searched for and pulled up into the incision. The organ should be brought entirely out through the opening or at least far enough to facilitate easy handling. The abdominal opening is packed with sterile gauze to stop any escape of stomach contents from entering the peritoneal cavity.

At this stage of the operation we will notice that the stomach is filled with gas and care should be exercised in opening it, lest the inward pressure expel the contents, thereby contaminating all surrounding tissues. To prevent this, it is well first to puncture the stomach with a hollow needle and let the gas escape.

Now the incision may be made after first locating an area with the least amount of blood supply. This is generally somewhere on the long axis of the stomach. Needless to say, if a foreign body is to be removed, it should be located, if possible, before making the incision, so as to save much time, and also give the operator an idea as to the size of the incision to be made. When this incision is made through the stomach, a small artery forceps is placed on each side and held outwardly by an assistant, similar to holding the laparatomy incision open. After the foreign body is removed, the edges of the stomach wound are swabbed with sterile gauze and painted with tincture of metaphen. The serous coats are drawn together with a No. 9 twisted silk, using a straight needle.

The method of suturing the stomach is extremely hard to explain on paper, yet it is of the utmost importance. We must close our incision so that it will be absolutely water-tight and we must be as quick as possible in doing so. For these two reasons the writer has adopted the modified continuous Lembert suture. A double row of these sutures is placed, thus inverting the serous coats twice and making the incision absolutely water-tight and, best of all, there is only one knot to tie. When the ends of this suture are pulled up, the silk is completely buried and generally the knot itself likewise is buried. The stomach incision having been closed, it is again painted with tincture of metaphen and returned to the abdominal cavity.

The forceps attached to the abdominal wall are now picked up by the assistant and the entire cavity is filled with liquor hexylresorcinol. The abdominal incision is closed in the usual manner. The peritoneum and muscle are sutured with a chromic

catgut No. 3 and the skin with a cotton or linen cord. No bandage is placed on the animal if it is to stay in the hospital. If it is to go home, then a many-tailed bandage is used.

Enterotomy

The technic for an enterotomy in many ways is similar to the foregoing. No duplication of description will be made. The landmark for this operation is posterior to the umbilicus. Starting one to two inches back of the umbilicus, it is carried for three or four inches, depending upon the size of the patient and the object in the bowel.

The bowel obstruction should be located, if possible, by palpation, so as to aid the operator to locate it more quickly when laparatomy is performed. The patient and operative field are prepared as for gastrotomy and the incision through the abdominal wall is made similarly. The bowel lesion is located and brought out through the incision. Sterile packs are placed around the protruding organ. The intestines should be ligated or clamped off a short distance from the injury. I prefer an ordinary forceps with rubber tubing slipped over the jaws; this prevents injury to the bowel. The incision may be made on either side of the foreign body (preferably in front), so as to get into as healthy tissue as possible.

In making the incision into the bowel, most of our text-books tell us to cut lengthwise of the organ, but I find it much better if the incision is made crosswise. By so doing, the bowel will heal, having only another "kink," while if made longitudinally the intestinal lumen will be greatly diminished and probably would lead to further obstruction in the future.

The foreign body is removed and the incision painted with tincture of metaphen and closed as in gastrotomy. The bowel is returned to the abdominal cavity. A good quantity of liquor hexylresorcinol is poured in and the abdominal walls closed in the usual way.

Cystotomy

Cystotomy generally is performed for the purpose of removing calculi. The landmark for this operation is that area directly in front of the penis in the male and in front of the edge of the pelvis in the female. The incision is made on the median line. The animal is prepared and laparotomy performed in the usual way. The bladder is drawn through the incision, after first having been emptied of the fluid content. The incision is made in a region where blood-vessels are fewer and then

the calculi removed. The bladder incision is sutured as for gastrotomy. The bladder is returned to the abdominal cavity and the wound closed as usual.

As to the indications for these operations, we will first consider gastrotomy. Probably the most frequent use for this operation is for the removal of foreign bodies from the stomach. Frequently the owner sees his animal swallow an object, in which case our diagnosis is rather easy. But in many cases we do not have that information. All too frequently the animal is not worth an x-ray examination. In these cases one must draw his conclusions from careful observation and palpation. If there is a foreign body in the stomach, we nearly always get a persistent vomiting a short while after eating. The animal is not able to keep sufficient food in the stomach and, as a result, becomes very emaciated. By careful palpation when the patient is in this thin condition, the object can often be felt. If all of these symptoms fail, one is justified in doing an exploratory laparotomy.

Ulcers of the stomach may be handled by taking out an elliptical piece of the stomach wall which contains the diseased tissue. This form of treatment is very successful if one is correct about his diagnosis.

Foreign bodies in the lower esophageal region may be removed easily by way of an opening into the stomach and reaching into the esophagus with long forceps. Care must be exercised lest either of the two branches of the pneumo-gastric nerve be injured, as they pass through the diaphragm above and below the esophagus.

Acute obstructions of the bowels may be relieved with enterotomy, if performed soon enough. The longer the operation is delayed, the weaker the patient and the greater the shock. If the animal shows a toxemia, it should be given intravenous injections of normal salt solution with 50 per cent glucose added.

In removing calculi from the urinary bladder one should not delay for too long a period, as the bladder wall becomes greatly thickened and healing is much harder to obtain. If an x-ray examination is not practical, one may be able to arrive at a diagnosis by feeling the calculi and by observing the urine. The patient usually passes blood and sometimes granules of a calcareous nature. In the female it is fairly easy to pass a metal sound and feel the calculi.

The patient should be fed a liquid, easily digested diet for four or five days after the operation. It is surprising, however,

to know how soon solid food can be taken with complete ease after a gastrotomy. One should hospitalize these cases, if possible. Home cases seem to have too much freedom and healing is slower.

In conclusion the writer would like to encourage practitioners to perform these operations more often. Acquainting themselves with the symptoms of foreign bodies and calculi will greatly lessen the mortality of these cases, as it permits the operation to be performed before the patient is in a weakened and toxic condition.

DISCUSSION

DR. E. J. FRICK: There are two little points I would like to bring out in discussing Dr. Bower's excellent paper. One of them is that in operating on the stomach, be sure that your dog is completely anesthetized. Otherwise, you may start a series of vomiting contractions.

It is a well-known clinical fact that many of these cases, for some reason that we do not understand, will improve following the laparotomy. There seems to be some connection with the sympathetic nervous system, and opening up the abdominal cavity in some of these obscure digestive disturbances and not doing any particular operation on the intestinal tract, will start the animal toward recovery immediately.

I believe that the average small-animal man is overlooking possibilities when he does not perform laparotomies in these cases of digestive disturbance.

DR. H. J. MILKS: I would like to call your attention to the new gastrointestinal sutures. The needle of these is so welded or attached to the catgut that the suture forms one continuous string. Since the needle is slightly larger than the catgut and there is no doubling of it, there is much less laceration of the tissues than with the ordinary needle, and much less danger of the stitch pulling or tearing out. You can secure silk fixed in the needle in the same way. The kind we use is made by Johnson and Johnson. These sutures do not cost much, and I believe they are a great help. We use them in all gastrointestinal work, and in suturing the uterus after a cesarean.

One other point: Be careful of your strong antiseptics in contact with peritoneal surfaces on account of the liability of adhesions. Do not use iodin around peritoneal surfaces because it is almost bound to cause adhesions.

DR. A. A. HERMANN: Where an x-ray is not available to the practitioner, it seems to me that the temperature of the animal is a very valuable aid in the diagnosis.

In my experience, with the presence of a foreign body, unless lacerations of the lining of the stomach or esophagus have occurred, there is no rise in temperature, whereas in the infectious diseases there usually is a marked rise.

Another distinguishing feature is the fact that an animal with a foreign body usually continues to have a good appetite, whereas an animal with an infectious disease does not. In the one case, the vomiting follows the food within five or ten minutes, and, in the other case, vomiting continues regardless of when the food, or what kind, is taken.

DR. A. R. THEOBALD: In the few cases of laparotomy that I have done, I have found that gastrotomy was more successful than enter-

otomy in its results. Often, after the foreign body is determined to be in the intestinal tract, it seems that the wiser thing is to attempt to get it into the stomach and remove it from the stomach, rather than the bowel.

I have had much better results by removing foreign bodies through the stomach wall rather than through the intestinal wall.

DR. E. E. WEGNER: I would like to ask Dr. Bower why he prefers silk rather than catgut.

DR. BOWER: The only reason why I prefer silk is because it is finer and a little more pliable, and when using a straight needle, to which I have become accustomed, it is just a little easier. It takes care of itself just as well as the catgut. I do think, however, that the catgut would do just as well if one is accustomed to using it. It is just a case of what the operator is accustomed.

I would like to emphasize the point brought out by Dr. Frick as to complete anesthesia. You are likely to get an intense vomiting, which will cause a lot of distress and embarrassment to the operator, if the patient is not anesthetized completely.

Commerce Department Continues Services to Drug Trade

Services to the drug trade will be continued in 1934, according to C. C. Concannon, Chief of the Chemical Division, Bureau of Foreign and Domestic Commerce, Washington, D. C. These services consist of three export statements, three import statements, one of imports for consumption, all published monthly, and a weekly bulletin which gives news of the world affecting the drug trade of the United States.

The export publications cover shipments of medicinal oils, biologics, pharmaceuticals, proprietary medicines, toilet preparations, crude drugs, etc. Import publications detail receipts of crude drugs, miscellaneous chemicals and perfumery. All these statements show quantity and value as well as countries of origin and destination. The statement of imports for consumption covers the entire field of drugs, chemicals, dyes, plastics, matches, etc. The weekly "World Trade Notes," issued each Monday, contains an average of 40 news items gathered from all parts of the world, together with a list of foreign trade opportunities received during the preceding week.

Mr. Concannon will be pleased to forward free sample copies of any of the Division's publications to anyone who is interested.

If an Englishman has been convicted of cruelty to his dog, he is prevented by the law of his country from ever owning or having the custody of another.

STUDIES ON A HERD INFECTED WITH BRUCELLA ABORTUS*

I. A Study of the Effects of Vaccination and of Segregation on the Control of Brucella Abortus Infection

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The recognition of a case of undulant fever at the Metropolitan Life Insurance Company Sanatorium, in 1926, led to a study of the Sanatorium patients and of the Sanatorium herd. The findings and the progress made in our studies up to 1929 have been presented in previous reports from this laboratory.[1,2] The first attack on the problem was through the examination of the herd for the presence of blood-serum agglutinins. All cows which showed agglutination titres of 1:120 or higher were segregated; those animals in which there was a titre of less than 1:120 were placed in a doubtful group. The second attack was a careful examination of the milk of all the cows. This study revealed that a number of cows were discharging *Brucella abortus*. Their milk was pasteurized. The milk of the remaining cows in the segregated group was pasteurized after each freshening and this procedure was continued unless a laboratory examination was negative.

Plans were put into effect to obtain an abortus-free herd by the gradual elimination of all reactors and by the building up of the herd with non-reactors of our own raising. When our studies began, the herd had already been vaccinated. We present here our findings with regard to the effects of vaccination and of segregation upon: (1) the abortion rate, (2) the presence of agglutinins in the vaccinated animals, and (3) the persistence of agglutinins.

The Sanatorium herd records show that for the years 1917-1920 there was an abortion rate of 19 per cent. At the suggestion of Dr. Cassius Way, vaccination with *Br. abortus* cultures was begun in November, 1920. During the next three years, the majority of the cows were vaccinated at least 60 days before breeding. In 1924, it was decided to limit the vaccination to heifers. In April, 1924, vaccination was discontinued. Table I shows that abortions were apparently under control during the years from 1922 to 1924. During this period there were but

*From the Hegeman Memorial Research Laboratory and the Farm of the Metropolitan Life Insurance Company Sanatorium. Received for publication, May 1, 1933.

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few unvaccinated animals in the herd. Early in 1925, in order to avoid delay before breeding, a group of young unvaccinated heifers was bred. The table shows that, in 1925, the abortion rate among these young unvaccinated animals was higher than for the herd in the year 1920. This led to the resumption of the use of vaccine. Six of the ten abortions occurring in 1926 were in newly imported stock. The significance of this will be discussed later in the paper. Vaccination was discontinued in June, 1926, and has not been used since.

It is of interest that there were many more retained placentas among the vaccinated cows than among the unvaccinated. The significance of this is but little understood.

The table suggests that vaccination can aid in the control of abortion. It also suggests that serious consequences may develop if this form of therapy is discontinued and fresh, unvaccinated stock is added to the herd.

TABLE I—*The number of pregnancies, abortions and retained placentas from 1917 to 1926.*

	UNVACCINATED			VACCINATED			TOTAL HERD		
	PREG.	ABORT.	R. P.	PREG.	ABORT.	R. P.	PREG.	ABORT.	R. P.
1917	22	4	—				22	4	—
1918	23	5	—				23	5	—
1919	47	5	3				47	5	3
1920	84	22	3				84	22	3
1921	54	5	11	23	2	2	77	7	13
1922	6	—	—	74	3	10	80	3	10
1923	3	—	—	79	2	11	82	2	11
1924	9	—	1	79	3	6	88	3	7
1925	16	5	1	64	1	11	80	6	12
1926	43	7	4	74	3	11	117	10	15
Total	307	53	23	393	14	51	700	67	74

Agglutination tests of the whole herd were done first in August, 1926. Of the 155 cows tested, 87 were found with titres of 1:60 or higher. Up to 1929, a total of nine agglutination tests had been made. Since then the herd has been tested twice a year.

Of the 259 cows tested from 1927 to 1931, 87 were vaccinated and 172 were unvaccinated. The agglutination titre of this series of animals against *Br. abortus* antigen is summarized in table II.

TABLE II—*The agglutination titres in the groups of vaccinated and unvaccinated cows from 1927 to 1931.*

	VACCINATED			UNVACCINATED		
	1:120 OR HIGHER	1:60 OR LOWER	NEG.	1:120 OR HIGHER	1:60 OR LOWER	NEG.
1927	53	22	12	13	12	40
1928	37	6	24	8	4	66
1929	24	9	14	3	1	85
1930	11	8	12	—	5	107
1931	5	6	6	—	1	113

We recognize that in 1927 there was a considerable amount of *Br. abortus* infection in the herd, while in 1931 there was practically none. It is therefore impossible to determine to how great an extent the presence of reactors was due to vaccination.

In the group included in 1927 there were 14 vaccinated heifers which had matured in 1926. It is significant that eleven of these had agglutinin contents ranging from 1:120 to 1:16,000, three had titres of 1:60, and not one was negative. Thirteen had developed agglutinins within three months after vaccination. Ten were tested before they were bred and nine of these showed agglutinins. In the 1927 group there were also 19 young unvaccinated cows which were running with the herd at large. Of these, three had agglutination titres of 1:120 or higher, four had titres of 1:60 and the remaining twelve were negative throughout. Ten were tested before being bred; all were negative but one which had a titre of 1:60. The comparison of these two groups is all the more significant since at this time the program of segregation had just been started and there was a considerable amount of infection in the herd.

Not only was there a marked contrast between the agglutinin content of the young animals in the vaccinated and unvaccinated groups in 1927, but agglutinins persisted in the blood of some of the vaccinated animals as long as they were under observation. The number of tests made on each vaccinated heifer varied from four to 16, making a total of 155 agglutination tests for the group. Within a year after vaccination, three of the heifers had become negative and these are the three which had agglutination titres of 1:60 at the beginning. Of the eleven heifers which had titres of 1:120 or higher, ten were in the herd two years after vaccination and at this time showed agglutination titres of 1:120 or higher. Four years after vaccina-

tion, five of the heifers were still reactors, three with titres of 1:120 or higher and two with agglutinin contents of 1:60.

Records of six of the 14 heifers were available five years after vaccination. The tests on this group are of interest. Two animals were negative. Each of these had a titre of 1:60 at the beginning and became negative within a year. One remained negative. The titre of the other has fluctuated between negative and 1:60. A third animal had an agglutination titre of 1:60. This animal had a titre of 1:120 at the beginning and had one negative test out of 16. Three animals had titres of 1:180. The original titres of these were 1:1,000 or higher and their lowest titre was 1:120. Whether the presence of agglutinins in these animals was due to vaccination or not it is impossible to determine. The fact remains, however, that agglutinins persisted for as long as five years after vaccination.

The group of young unvaccinated cattle in 1927 considered above gives the following data relative to the persistence of agglutinins. The three animals which had agglutination titres of 1:120 or higher were disposed of within two years after this study was begun. These animals always had titres of 1:120 or higher. The four animals with original agglutination titres of 1:60 never had titres higher than this but 14 testings of the sera gave readings of 1:60, while 28 were negative. Two of these animals were in the herd six years after our studies were begun and they are negative at present. The two animals have had 32 tests, eight at 1:60 and 24 negative. The remaining twelve have at no time shown a positive test. A total of 120 tests was done on these negative animals. Three of these animals were still in the herd six years after the studies were begun. They have had a total of 44 tests all of which have been negative. Many of the animals have been disposed of for one reason or another, their disposal having no reference to the agglutination tests.

When one compares the records of the persistence of agglutinins in the vaccinated and unvaccinated groups here considered, it appears that vaccination must have played a part in the positive agglutinations found. The records of two of the older cows are of interest in this connection. Cow 261, which is considered later in the paper, received her last dose of vaccine in December, 1921. She had never had a titre in excess of 1:120 but still maintained a titre of 1:60 in October, 1930, nearly nine years after vaccination. Cow 305 was given a single dose of vaccine in May, 1923. Her titre was over 1:1,000 in 1927, later

dropped to 1:540 and 1:180 but was still 1:180 in January, 1931, nearly eight years after vaccination.

It was noted in the records of agglutination tests done upon the herd that there was some fluctuation in the agglutinin content from time to time. Some of the reacting cows with the higher titres showed at times a decrease with a subsequent rise. Some of the animals, which had titres of 1:60, on later tests showed variations from negative to 1:60. Because of these findings we selected for more intensive study with regard to agglutinin content of the blood: (1) a group of animals that gave positive tests consistently, (2) a group which showed occasional reactions, and (3) a negative group. Twenty-two cows were selected. We wished to determine, if possible, whether there might be a certain period in the yearly cycle of the cow during which the agglutinin content was consistently higher than at any other period. In this way we felt it might be possible to ascertain the best time for determining reactors.

The tests, when possible, were started on each animal a few months before she was due to calve and continued monthly for a year or longer. At the close of the study it was found that, in nine animals, the agglutination titre was never less than 1:60; in six, there occurred fluctuations between negative and 1:60; and in seven no positive tests were obtained throughout the study. All of the animals in the first group and three in the second group had been vaccinated.

With the exception of two heifers, which were in their first pregnancy, all of the animals had calved once or more. One cow had aborted once and one had aborted twice.

Table III gives in detail the agglutination titres of all of these animals. The dates of calving, breeding, and end of the milk-production period are indicated approximately. The first nine animals are those which had been vaccinated and were consistent reactors. It will be noted that there is considerable fluctuation in the agglutinin content of some of the cows while others fluctuate but slightly. Cow 306 is of special interest because the titre fluctuates from 1:120 to negative and then to 1:60, with but three positive tests out of 17. The table shows that vaccinated cows 194, 362 and 379 and unvaccinated cows 306, 455 and 503 all had positive as well as negative tests during the course of these observations.

A careful study of the table shows that there is no definite relation between the time of year, calving period, breeding per-

TABLE III—*Monthly agglutination tests.*

Cow	Maximum Titre	Consistent Reactors																	
		1929				1930											1931		
		9–10	10–7	11–7	12–2	1–6	2–3	3–3	4–7	5–5	6–2	6–30	7–28	9–8	10–7	10–23	12–1	1–5	1–29	2–9
V261	60	.....	.....	120D	120	120	120	120C	60	60	60	60	60	60	60	.....	.....	.....	.....	.....
V305	8,000	540D	540	540C	180B	180	180	180	180	180	180	180	180	180	180D	60	180C	180	180	.....
V327	540	540D	180	180	120C	120B	120	120	120	120	120	120	120	180	180	180	.....	.....	.....	.....
V343	540	.....	540D	180	120C	120	120	120B	120	120	120	120	120	120	120	120	180D	180C	.....	.....
V364	180	.....	.....	120D	120	60	60C	60	60	120	120	60	120	120	120	60	.....	.....	.....	.....
V372	4,000	.....	180D	540	540	540C	540	540	540B	540	540	540	540	540	540	180	540D	540C	.....	540
V381	2,000	.....	.....	180	.....	.....	.....	180	180D	180	180C	180	180	180	180	180	180	180	180	.....
V389	16,000	.....	.....	120	.....	.....	.....	120	.....	.....	120D	120	180C	180	180	180	180	180	180	.....
V392	2,000	.....	.....	180	.....	.....	.....	180	180D	180C	180	180	120B	120	120	120	120	180	120	.....

Cow	Maximum Titre	Occasional Reactors																		
		1929				1930												1931		
		9–10	10–7	11–7	12–2	1–6	2–3	3–3	4–7	5–5	6–2	6–30	7–28	9–8	10–7	10–23	12–1	1–5	1–29	2–9
V194	0	.....	.....	S 60D	S/60	0	S 60C	0	0	S/60	S/60	S 60B	0	S/60	0	0	.....	.....	.....	.....
V362	180	.....	.....	60D	S/60	S 60C	S/60	0B	60	60	0	60	60	60	S 60D	60	60A	60	.....	60
V379	180	.....	60C	60	0	0B	60	S/60	60	60	60	60	60D	60	60	60C	.....	.....	.....	.....
U306	120	.....	120D	0	0C	0	0B	0	0	0	0	0	0	0D	0	0	60C	0	.....	60B
U455	0	.....	.....	0	0	0	0D	0	0	0C	0	S/60	S 60B	0	0	0	0	0	.....	.....
U503	0	.....	.....	0	0	0	0	0	0C	0	S/60	60B	S/60	0	0	0	0	0	.....	.....
		Non-Reactors																		
U269	0	.....	0D	0C	0	0B	0	0	0	0	0	0	0D	0	0C	0	0	0	.....	.....
U409	0	.....	0D	0	0C	0	0	0B	0	0	0	0	0	0	0D	0	0	0C	.....	0
U415	0	.....	.....	0D	0	0C	0	0B	0	0	0	0	0	0D	0	0	0C	0	.....	.....
U422	0	.....	C	0	0	0	0B	0	0	0	0	0	0	0D	0	0	0C	0	.....	.....
U443	0	.....	.....	0D	0	0	0C	0	0B	0	0	0	0	0	0D	0	0	0C	.....	0
U446	0	.....	.....	0C	0	0	0B	0	0	0	0	0	0	0D	0	0C	0	0	.....	.....
U502	0	.....	.....	0B	0	0	0	0	0	0	0	0	0C	0	0	0B	0	0	.....	.....

V = Vaccinated.
U = Unvaccinated.
S = Partial agglutination.

A = Aborted.
B = Bred.

C = Calved.
D = Dry.

iod, or the milking period, and the degree of the agglutinin content of the blood.

From the herd data, we were able to compare the effects of vaccination and of segregation on the abortion rate. The program of vaccination extended from 1920 to 1926 and the program of segregation, from 1927 on. The percentages of abortions and of retained placentas in the groups of non-vaccinated and of vaccinated cows were determined for each year. The graph in figure 1 gives these data for each year from 1917 to 1931. The high percentage of abortions from 1917 through 1920 led to the adoption of the program of vaccination. There was a decided drop in the percentages of abortions among the vaccinated cows from 1921 to 1924. The data on the unvaccinated group during this period are of little value because of the small number of animals. In 1925 there was a marked increase in the percentage of abortions in the herd. These abortions occurred among the unvaccinated heifers of our own raising. This experience led to the resumption of vaccination. There was a reduction in the percentage of abortions in 1926. In other

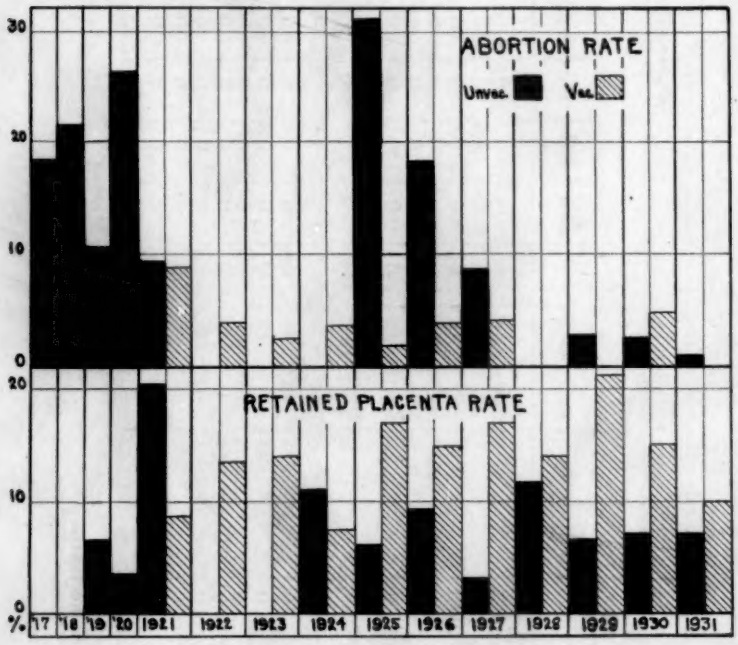

FIG. 1. Graph showing abortions and cases of retained placenta in vaccinated and unvaccinated groups from 1917 to 1931.

words, the graph demonstrates clearly that prophylactic vaccination in an abortus-infected herd was effective. There is, however, a disturbing factor in such a procedure in that the percentage of retained placentas is distinctly higher in the vaccinated groups than in the unvaccinated, with the exception of the year 1921. This phenomenon is noted long after vaccination had been discontinued, so that it appears that this therapy had some effect on the retention of placenta.

Beginning in 1926, the number of cattle in the non-vaccinated group rapidly increased, while the number in the vaccinated group decreased as the program of segregation replaced that of vaccination. It will be noted in figure 1 that the percentage of abortions in the unvaccinated group remained very low from 1927 on, as did also the percentage of retained placentas. In other words, when one compares the effect of vaccination with that of segregation on the control of abortion and of retained placentas, it is readily seen that an effective segregation without vaccination is preferable in the building up of a herd free from *Br. abortus* infection. The abortion rate for the year 1925 shows also that it is unwise to add fresh, unvaccinated stock to a vaccinated herd.

In 1919 and again late in 1925, stock purchased from outside was added to our herd. No data were available as to whether these two groups of animals were infected or were vaccinated prior to purchase. The high abortion rates in 1920 and in 1926 were due to the abortions in these groups. The question naturally arises as to whether the abortions were due to infection already present or to infection picked up subsequent to introduction into our herd. There are no conclusive data on this point. From these experiences it can be seen readily that it is an unwise procedure to add outside stock to an infected herd whether the herd is vaccinated or not.

Our studies were begun in 1927 with an infected, vaccinated herd. We have been able to follow the agglutinin content of the herd as a whole from this period through the program devised for obtaining a herd free from *Br. abortus* infection. We present in figure 2 a graph showing the distribution of the agglutination titres in the groups of vaccinated and unvaccinated cows for April, 1927, and for the year 1931.

A study of the 1927 graph shows that 78 per cent of the vaccinated and 37 per cent of the unvaccinated groups gave positive agglutination tests. When a comparison of the graph of 1927 is made with that of 1931, it is seen that 56 per cent

of the vaccinated and 17 per cent of the unvaccinated group had agglutination titres of 1:120 or higher in 1927, while there was none in 1931. A noteworthy feature of the effect of segregation on the unvaccinated group is the increase of negative tests from 63 per cent, in 1927, to 98 per cent, in 1931. This result is especially striking since practically all of the cattle of the herd of 1931 were unvaccinated, while in 1927 only 43 per cent were unvaccinated. When one considers the vaccinated group in these years, it is necessary to bear in mind that there were 85 of these in April, 1927, while in 1931 only twelve were present. The difference in the size of the two groups is due to the fact that the vaccinated animals were disposed of gradually, as the program of segregation was carried out. There is no agglutination titre higher than 1:60 in the 1931 graph because, some months before the entire herd was tested, all vaccinated cows with titres higher than 1:60 had been disposed of. When one compares the relative percentages of the frank negatives and the 1:60 reactors in 1927 and 1931, it is seen that the percentage of negative reactors has risen markedly. However, there has not been nearly so great a relative reduction in the positive reactors in the vaccinated group as there has been in

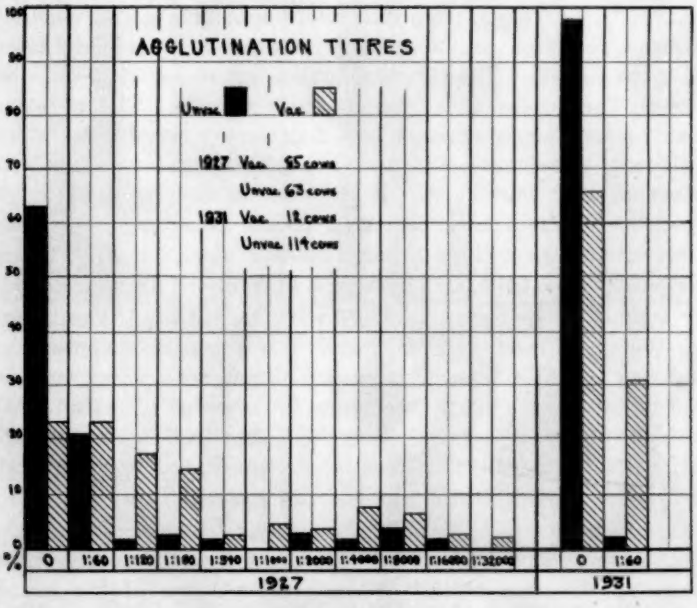

FIG. 2. Graph showing agglutination titres in vaccinated and unvaccinated groups, 1927 to 1931.

the non-vaccinated. Whether this is the effect of vaccination or whether it is the persistence of infection it is impossible to determine.

Discussion

The studies here recorded are not experimental studies but the outcome of a practical attempt to control abortions, presumably caused by *Br. abortus*, in a valuable dairy herd. Detailed herd records were available from 1917 on. Living abortus vaccine was used from 1920 to 1926. Serological studies were begun in 1926 and bacteriological studies a year later.

The use of living abortus vaccine in our infected herd served the purpose for which it was intended. Although the percentage of retained placentas increased, the abortion rate for the herd decreased, from 26.2 per cent, in 1920, to 3.4 per cent, in 1924. The reduction of the abortion rate subsequent to the use of living *Br. abortus* vaccine is in accordance with the findings of Stockman,[3] Hadley,[4] Schroeder,[5] Smith and Little,[6] Fitch and Boyd,[7] Hart and Traum,[8] and others.

Our records indicate that live vaccine therapy, once started, should not be abandoned without providing some safeguard for unprotected animals. The discontinuance of vaccine therapy without the segregation of the unvaccinated animals apparently caused the increase of the abortion rate in 1925. Smith and Little[6] and Lubbehusen *et al*[9] found that high abortion rates prevailed among the unvaccinated animals when vaccinated and unvaccinated cows were pastured together. Fitch *et al*[10] state that the use of vaccine must be continued from year to year.

Another marked increase in the abortion rate of our herd occurred in 1926, after the addition of some Canadian stock. Some of these animals were pregnant when they were received; none was vaccinated before being bred. Six out of 15 pregnancies in this group terminated in abortions. We do not know whether the animals were infected when they arrived or whether they acquired infection from our herd. Eichhorn and Potter,[11] Connaway *et al*,[12] White *et al*,[13] Barnes,[14] Kalkus and Sawyer,[15] Fritz and Barnes,[16] and others find an increase in the abortion rate the usual consequence of exposing susceptible stock to infected herds.

Vaccination, when used regularly, obviously brought about the desired decrease in the abortion rate in our herd, but this was accompanied by an increase in the percentage of retained placentas. This rate remained high for five years after vaccination had been discontinued.

The effect of vaccination with living *Br. abortus* vaccine upon the production of serum agglutinins in cows is shown strikingly by our herd records. Fifty-three (61 per cent) of the vaccinated cows in the herd in 1927 had titres ranging from 1:120 to 1:32,000; and 22, or an additional 25 per cent, had titres of 1:60. Only thirteen (20 per cent) of the unvaccinated cows had titres of 1:120 or higher and twelve (18 per cent) had titres of 1:60. Vaccinated heifers showed agglutinins in less than three months after vaccination and before being bred. Unvaccinated heifers showed very little tendency to form agglutinins either before or after breeding.

The vaccinated cows were characterized not only by the prompt appearance of agglutinins but also by the persistence of agglutinins in the blood serum. A few in the 1:60 group eventually became and remained negative but this was not true of those with a higher titre. Although there was some decrease in titre, agglutinins were demonstrable as long as the animals were under observation, one to nine years after vaccination. Buck and Creech,[17] Hadley and Welsh,[18] and Hart *et al*[19] report the prompt appearance of agglutinins after vaccination or experimental infection. They report also, in general, markedly decreased or negative titres within six months to two years. Smith and Little[6] and Lubbehusen *et al* [9] report the persistence of fairly high titres, in some instances for three or four years after vaccination. Birch and Gilman,[20] Clark,[21] Huddleson and Smith,[22] and Newsom and Cross[23] report the persistence of agglutinins in infected animals in herds under observation for from two to eight years. Few investigators have had an opportunity for obtaining detailed records of herds with long established high abortion rates and vaccination programs. This may account for the lack of data on the persistence of agglutinins in vaccinated animals for such long periods as we have reported here.

The data on other vaccinated herds might suggest that the high and persistent titre of some of our vaccinated animals is evidence of natural rather than induced infection. In our herd no attempt to segregate reactors was made prior to 1927 and there were very few reactors of high titre among the unvaccinated stock. This suggests that vaccination rather than natural infection might have been an important factor in the persistence of infection in the herd.

Monthly serological tests for a period of a year or more on 22 cows did not indicate that the time of year, calving period,

breeding period or milking period could be correlated with increase or decrease of agglutination titre of the reacting cows or heifers tested. There were variations in titre from month to month. The monthly tests on this group did not indicate any one time in the year when the greatest number of reactors might be found. A single negative test was no guarantee that subsequent tests would be negative. We did not observe in this small group of animals a consistent decline in agglutinin content near the time of calving, as has been reported by Barnes[14] and by Rettger *et al.*[24] The data on monthly agglutinations given in the tables of Hart and Traum,[8] Huddleson and Smith,[22] and Birch and Gilman[25] indicate no evidence of decrease or increase of titre associated with calving.

We have found vaccine therapy of value in the control of abortions but not in the control of infection. For the period that our herd has been under observation, the segregation of reactors and the raising of our own stock has been more effective than vaccination, not only in the control of abortions, but in the elimination of *Br. abortus* infection. There were only six abortions in 385 pregnancies from 1928 to 1931 and there were no cows in the herd with more than partial reactions at 1:60 in the 1931 or the 1932 testings. Our observations are in accord with the concensus of opinion, as voiced by Lubbehusen *et al*,[9] Fitch *et al*,[10] Barnes,[14] Kalkus and Sawyer,[15] Graham *et al*,[26] Simms *et al*,[27] Rettger *et al*,[23] and others, that the best way to control *Br. abortus* infection is on the basis of a clean herd determined by systematic testing, and that segregation is more effective than vaccination in establishing such a herd.

Eichhorn and Potter,[11] White *et al*,[13] Barnes,[14] Turner,[29] and others report the disastrous effects of lack of segregation in the control of infectious abortion. We have in our records three rather striking examples of this. In 1919, a herd of 50 cows was purchased outside. Of the 21 newly acquired cows which calved that year, only one aborted. In the following year, 47 of the cows in the group were pregnant and of these 18 aborted.

In April, 1924, when the abortion problem seemed to be under control, vaccination was discontinued but no attempt was made to separate the vaccinated and unvaccinated cows. A marked increase in abortions in the unvaccinated stock in 1925 was the direct result. In the fall of 1925, the program of vaccination was resumed.

The third example occurred when 15 new cows from Canada were added to the herd in 1925. At this time no attempt to vaccinate or to segregate was made. The high abortion rate in 1926 is due to abortions in the Canadian group.

We thus have instances of the serious consequences of (1) adding outside stock to an unvaccinated herd with a high abortion rate, (2) of adding our own unvaccinated young stock to our vaccinated herd, and (3) of adding outside stock to a vaccinated herd.

We have no serological data covering these periods and no knowledge as to whether the cows introduced in 1919 and in 1925 were infected or had been vaccinated previously. If they were infected when purchased, they showed themselves as susceptible to infection as the young unvaccinated stock of our own raising. If the Canadian cows did bring fresh infection into our herd, the success of the program of vaccination in 1926 and of segregation in the years following is remarkable.

The effect of segregation on agglutination titre is almost as striking as its effect upon abortion rate. With the gradual eliminations of the reactors of high titre and the segregation of the remainder, titres gradually declined from year to year. The young stock which is being added to the herd constantly is apparently free from infection. In 1931, 96 per cent of the cows in the herd were negative. The remaining 4 per cent had titres of not more than 1:60 and these were segregated.

CONCLUSIONS

The data presented were obtained from a practical attempt to control *Brucella abortus* infection in a valuable dairy herd. From these data the following conclusions are drawn:

1. Vaccination with living abortus bacilli decreased the percentage of abortions in the herd but increased the percentage of retained placentas.

2. Segregation was found more effective than vaccination in the control and elimination of infectious abortion. Vaccination without segregation was not successful in the elimination of the infection.

3. Agglutinins were demonstrated in the blood serum of 86 per cent of the vaccinated cows tested in 1927. Agglutinins persisted in the blood as long as the animals were under observation. In one case this was nine years after vaccination. Occasionally, vaccinated reactors with maximum titres of 1:120 became negative. This was not true of vaccinated cows with higher titres.

4. Monthly serological tests failed to show any correlation between time of years, breeding period, calving period or milk-production period and an increase or decrease in agglutination titre. Tests made on the herd at one time were as significant as tests made at any other time. Repeated tests are essential to insure the maintenance of a negative herd.

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New Facts Simplify Bot Control

The control of bots in horses is now much more effective as a result of recent experiments of the Bureau of Animal Industry and the Bureau of Entomology of the U. S. Department of Agriculture and the development of campaigns to rid whole communities of large numbers of the parasite in one season.

In the past, many horses have been treated for bots too early in the season to get all of the parasites. It is now known that the larvae of the common bot, hatched from eggs on the hairs of the animal, penetrate the surface of the tongue and mucous membranes of the mouth and stay there from 20 to 28 days before migrating to the stomach. Now, instead of giving the treatment immediately after the first freezing weather, it is plain that the treatment should be delayed for a month after a period of freezing weather lasting a day or longer. Treatment too early fails to get the bots that have not yet reached the stomach. Freezing weather kills the adult flies, but the immature bots in the tongue, or on the way from the tongue to the stomach, escape the freeze on the outside and the chemical treatment on the inside if the latter is given too early.

The following procedure is suggested: After the first freezing weather lasting a day or more, destroy all bot eggs on the hairs of horses by washing the parts with a 2 per cent solution of cresol or a suitable dilution of a reliable coal-tar creosote dip; wait a month for the bots in the horse to concentrate in the stomach and then give the treatment. This may be given any time during the winter before March 1, but the earlier it is done, the better for the horses.

Experiments indicate that liquid carbon disulfid is the most effective agent available for the destruction of bots in the stomach of the horse. Tests with the so-called "mass" carbon disulfid capsules, in which the drug is adsorbed in a powdered base, indicate that these capsules are not so effective as are capsules containing the liquid drug. Furthermore, the mass capsules become brittle with age and there is a considerable loss of carbon disulfid.

STUDIES IN HOG CHOLERA IMMUNITY*

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Since the discovery by De Schweinitz and Dorset, in 1903, that hog cholera is due to a filtrable virus and that immunity against this disease may be induced in a susceptible hog by the administration of serum from an immune hog and the virus from a sick hog, a great number of experiments have been carried on by various investigators to gain additional knowledge relative to this means of producing immunity against this disease, especially through the vaccination of young pigs by using serum and virus (simultaneous method) and by using virus only.

Reynolds[1] reported that young pigs from immune sows were found to have a high grade of immunity, while those from susceptible sows were found to have practically no resistance against cholera. The same investigator stated that the temporary immunity in young pigs from immune mothers lasted about five weeks and could be made permanent by administering a small dose of virus during the period of immunity.

Benner[2] showed that the temporary immunity of the offspring from immune sows is acquired either *in utero* or by the colostrum, or by both, and not by the milk.

Benner[3] states that the use of virus alone is too dangerous to become practical as a means of producing permanent immunity in hogs.

Pickens *et al*[4] states that the temporary immunity is not made comparatively permanent in all cases of inoculation with simple unmitigated virus, if given during the early period.

The objects of this experiment were: (1) to determine, if possible, if pigs born of cholera-immune sows could be immunized successfully and permanently against hog cholera by being artificially inoculated with virus during the nursing period of their lives, (2) to determine if the sows immunized in this manner would confer temporary immunity to their offspring, and (3) to determine as far as possible the permanency of the immunity, and, incidentally, to observe what effect, if any, such a method of immunization would have upon the fecundity of the sows.

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Method of Procedure

On July 18, 1924, a number of susceptible gilts were purchased and vaccinated with the simultaneous method, and later bred. When these gilts farrowed, the young pigs were inoculated artificially with a small quantity (1 to 2 cc) of hog cholera virus. As a rise in temperature is one of the principal symptoms of hog cholera, the temperatures of these pigs were taken before inoculation and for several days after inoculation. Some weeks (12 to 15) later, the immunity of the male pigs was tested by reinoculating them with 5 cc of hog cholera virus. The temperatures of these pigs were taken before receiving the virus and for several days thereafter. If these pigs failed to become sick they were disposed of, and it was assumed that the gilts were likewise immune to hog cholera. When old enough, these gilts were bred to produce offspring with which to continue the experiment. The plan at first was to carry on the experiment by using the first litter only.

The number in each litter is stated to show the fecundity of the sow. The difference in the number born and the number inoculated is due to the usual causes of mortality among young pigs. It was thought inadvisable also to inoculate the weakly and cripple pigs, those that would probably die from other causes within a few days after inoculation.

After the pigs were weaned, the sow was to be disposed of. Some of the sows were hyperimmunized and made excellent donors of anti-hog cholera serum.

Owing to a number of factors, such as abortion, cannibalism, lying upon the little pigs, disadvantages of having the sows farrowing at the serum plant, a preponderance of male offspring and many other causes, the experiment did not proceed satisfactorily at first. In November, 1927, only two sows remained with which the experiment could be continued. These sows were farrowed in September, 1926, and had farrowed their first litters in October, 1927.

Results

The results from the vaccination of young pigs were rather encouraging and in spite of the poor showing from other causes it was decided to modify the management somewhat and continue with the work, using these two sows which were of the second generation.

For convenience these sows are designated as "A" and "B." Sow "A" (second generation) farrowed her second litter (third generation) on May 17, 1928. This litter consisted of 11 pigs

—three sows and eight boars. When 16 days old, seven of this litter, two sows and five boars, were inoculated with 2 cc of hog cholera virus, made and used by the Station serum plant. The results of this test are given in table I.

TABLE I—*Temperatures of pigs from sow "A" inoculated with hog cholera virus.*

PIG	VIRUS (CC)	PRE- AND POST-INOCULATION TEMPERATURES										
		*	1†	2	3	4	5	6	7	8	9	10
1	2.0	3.6‡	2.8	3.9	3.9	4.2	4.0	4.0	3.8	3.8	3.6	3.8
2	2.0	3.0	3.0	3.8	3.0	4.2	4.0	4.0	3.8	3.2	3.2	3.0
3	2.0	3.8	2.8	4.0	3.8	3.0	3.8	3.2	4.2	3.6	3.0	3.0
4	2.0	3.5	2.6	2.2	2.4	2.6	2.6	3.0	3.0	3.8	3.2	3.0
5	2.0	3.6	3.8	3.8	3.9	4.0	4.0	4.2	6.0	3.2	3.2	3.0
6	2.0	4.0	3.5	3.8	4.0	4.0	4.0	4.0	3.8	3.4	3.0	3.0
7	2.0	3.0	3.5	4.0	4.0	4.0	4.0	3.8	3.0	3.0	3.0	3.0

*Pre-inoculation temperatures.
†First day after inoculation.
‡Degrees above 100°F.

Seventeen days after inoculation, one male pig died. The cause reported by the Department of Pathology, based on an autopsy, was anemia. These pigs were weaned at the age of nine weeks and five days. Fifteen weeks after the first inoculation of hog cholera virus, the four remaining male pigs were inoculated each with 5 cc of hog cholera virus obtained from a commercial firm (see table II). The females were bred. The old sow was rebred, but failed to conceive and was sold May, 1929, weighing 565 pounds.

TABLE II—*Temperatures of pigs* 3, 4, 5 *and* 7 *(table I)* following inoculation with virus.*

PIG	VIRUS (CC)	PRE- AND POST-INOCULATION TEMPERATURES										
		†	1	2	3	4	5	6	7	8	9	10
3	5.0	4.0	4.2	4.3	2.8	3.2	4.2	3.6	4.6	3.4	3.4	2.2
4	5.0	4.0	3.7	4.9	2.6	3.4	2.5	3.0	2.4	2.6	2.9	2.5
5	5.0	3.6	4.2	3.6	2.5	3.7	4.3	4.0	4.0	4.0	4.5	2.8
7	5.0	3.6	3.4	3.1	3.0	2.2	1.9	2.8	2.8	2.2	2.2	2.6

*Pig 1 (sow 741) and pig 2 (sow 742) were not reinoculated. These were used to continue the project. Pig 6 died on June 19, of malnutrition.
†Pre-inoculation temperatures.

Owing to the similarity in many of the temperature charts, only those which are deemed of interest are given.

Sow "B" (second generation) farrowed her second litter (third generation) on May 18, 1928. This litter consisted of 12 pigs—seven females, four males, and one dead. When 15 days old, four females and four males each received 2 cc of hog cholera virus, and the temperatures were taken for ten more days. The pigs were weaned when nine weeks and five days old. The four males each were reinoculated with 5 cc of hog cholera virus 15 weeks and four days after the first inoculation.

The pre-injection temperatures of these pigs before inoculation ranged from 103 to 105° F. The temperature on the fifth day ranged from 104 to 105° F. The greatest increase for any one pig from date of inoculation was 1.6° F. The temperatures on the tenth day varied from 103 to 104° F. All appeared healthy throughout this time. The highest temperature shown following reinoculation was 104.4° F. on the fourth day. This pig showed an increase of 1.2° F. above that recorded on day of reinoculation.

Sow "B" was rebred and farrowed her third litter (third generation) on April 16, 1929. This litter consisted of nine pigs. When 18 days old, seven received each from 0.5 to 1.5 cc of hog cholera virus. These pigs were not thrifty, hence the small dose of virus administered. One was reinoculated 19 weeks after the first inoculation. The results are given in tables III and IV.

TABLE III—*Temperatures of baby pigs from sow "B" inoculated with hog cholera virus.*

PIG	VIRUS (CC)	PRE- AND POST-INOCULATION TEMPERATURES										
		*	1	2	3	4	5	6	7	8	9	10
1	1.25	2.6	2.2	....	3.2	0.2	2.9	1.4	4.0	3.0	†	.....
2	1.50	3.0	3.9	3.2	1.8	1.3	3.0	3.6	2.5	2.1	2.8	5.1
3	1.25	2.0	3.0	3.0	0.2	1.6	3.1	3.6	1.7	1.6	1.9	1.0‡
4	1.50	4.0	3.1	3.0	3.2	2.2	3.4	3.5	2.1	1.9	1.6	1.4§
5	0.50	1.0	1.2	....	0.5	0.0	3.5	3.5	1.1	3.0	.....	2.5
6	1.00	2.0	2.6	1.0	1.0	1.1	3.6	1.6	1.3	0.0	1.4	5.0
7	1.00	1.4	3.0	2.2	3.1	1.3	2.9	5.3	5.0	3.5	†	.....

*Pre-inoculation temperatures.
†Eaten by sow.
‡Died of pneumonia, August 2, 1929.
§Destroyed, May 14, 1929.

Sow 741 (third generation), offspring of Sow "A," born May 17, 1928, farrowed first litter (fourth generation) on April 3, 1929. There were six in the litter. Four pigs were inoculated

TABLE IV—*Temperatures of pig 2 (table III) reinoculated with virus.*

PIG	VIRUS (CC)	PRE- AND POST-INOCULATION TEMPERATURES								
		*	1	2	3	4	5	6	7	8
2	5.0	2.4	2.2	2.3	3.0	3.5	2.5	2.4	2.6	2.3

*Pre-inoculation temperature.

each with 1.0 to 1.5 cc of hog cholera virus when ten days old. Of these four that were inoculated, one was eaten by the sow on the eighth day, one died of malnutrition after the 13th day, and the two others died on the 32nd and 39th day respectively. Postmortem examination showed a ruptured liver in each case.

Sow 741 farrowed her second litter of eight pigs (fourth generation) on September 6, 1929. Three of the pigs were inoculated each with 2 cc of hog cholera virus when five days old. One pig was reinoculated with 5 cc, 135 days after the first inoculation. The results are given in tables V and IX.

TABLE V—*Temperatures of pigs from sow* 741 *inoculated with hog cholera virus.*

PIG	VIRUS (CC)	PRE- AND POST-INOCULATION TEMPERATURES							
		*	2	3	5	6	7	8	9
1	2.0	3.8	3.5	3.7	4.6	6.0	3.6	3.4	3.5
2	2.0	2.2	4.2	4.7	4.9	6.0	4.8	4.1	4.8
3	2.0	2.0	3.5	3.7	4.6	3.9	3.6	2.5	3.4

*Pre-inoculation temperatures.

Sow 742 (third generation), same litter as 741, farrowed first litter (fourth generation) on August 28, 1929, with 11 in the litter.

Eight of the pigs were inoculated with 2 cc of hog cholera virus when 14 days old. The temperatures on the day of inoculation varied from 102 to 103.5° F. On the fifth day, the temperatures ranged from 102.5 to 104.9° F. The greatest increase was 2.9° F. All of the pigs appeared healthy, except one that died of malnutrition on the sixth day. Its temperature on the day previous to its death was 102.5° F.

One hundred and thirty-five days after inoculation, one was reinoculated with 5 cc of virus. The results of reinoculation are given in table IX.

Sow 742 farrowed her second litter (fourth generation) on May 4, 1930, with 14 in the litter. Five of the pigs were inoculated each with 1.5 to 2.0 cc of hog cholera virus when 16 days old. The results are given in table VI.

Owing to the termination of the experiment, none of the pigs from sow 742 (second litter), sow 744 (second litter) and sow 746 (second litter) were reinoculated.

TABLE VI—*Temperatures of pigs from sow 742 inoculated with hog cholera virus.*

PIG	VIRUS (CC)	PRE- AND POST-INOCULATION TEMPERATURES										
		*	1	2	3	4	5	6	7	8	9	10
1	1.5	2.2	3.0	2.8	3.6	3.2	3.6	3.4	3.0	3.0	3.2	3.6
2	1.5	2.4	2.6	3.4	5.0	3.8	5.2	4.6	4.2	4.4	4.0	4.2
3	1.5	3.0	4.2	3.8	4.2	4.8	4.4	3.6	3.4	3.0	3.0	3.4
4	2.0	1.4	3.4	3.2	3.8	3.8	3.6	3.0	2.8	2.8	3.0	3.0
5	2.0	3.2	3.4	3.2	4.0	2.6	2.4	2.8	2.8	3.0	3.6	4.0

*Pre-inoculation temperatures.

Sow 743 (third generation), offspring of Sow "B," born May 18, 1928, farrowed her first litter on April 22, 1929, with seven in the litter. Five of the pigs (fourth generation) were inoculated each with 1.25 to 1.5 cc of hog cholera virus when 12 days old.

The pre-injection temperatures of these five pigs varied from 102 to 102.8° F. At no time during the ten days following the inoculation did the temperature of any pig rise above 103° F. On the fifth day, the range of temperatures was from 100.5 to 102.5° F.

One of the above-mentioned five was reinoculated 141 days after the first inoculation. The pre-injection temperature was 102.5° F., the highest post-injection temperature was 103.4° F. on the third day.

Sow 743 farrowed her second litter (fourth generation) on December 17, 1929, with ten in the litter. Three of the pigs were inoculated each with 2 cc of hog cholera virus when 14 days old. These pigs were kept on a farm and no post-inoculation temperatures were taken. The pigs apparently were healthy from the time of inoculation until three weeks later, after which no further attention was given to them.

Sow 744 (same litter as 743) farrowed her first litter on September 2, 1929, eight in the litter (fourth generation). Six pigs received each 2 cc of hog cholera virus when nine days old.

One was reinoculated with 5 cc of virus, 135 days after the first inoculation.

TABLE VII—*Temperatures of pigs from sow 744 inoculated with hog cholera virus.*

PIG	VIRUS (CC)	PRE- AND POST-INOCULATION TEMPERATURES							
		*	2	3	5	6	7	8	9
1	2.0	4.0	4.3	3.5	4.5	4.3	4.0	4.6	4.9
2	2.0	4.2	4.3	4.3	4.7	4.2	3.8	4.0	4.0
3	2.0	4.0	4.3	4.2	4.6	4.0	3.4	3.9	3.7
4	2.0	1.5	4.2	4.2	4.5	3.8	3.4	3.7	3.6
5	2.0	2.0	3.8	3.9	4.4	3.8	3.1	3.0	3.5
6	2.0	2.2	3.2	3.5	4.1	3.7	3.0	2.6	3.2

*Pre-inoculation temperatures.

Sow 744 farrowed her second litter on March 6, 1930, seven in the litter (fourth generation). Four pigs were inoculated each with 2 cc of hog cholera virus. One received 1.5 cc when 15 days old. Results of the inoculation are given in table VIII.

TABLE VIII—*Temperatures of pigs from sow 744 inoculated with hog cholera virus.*

PIG	VIRUS (CC)	PRE- AND POST-INOCULATION TEMPERATURES													
		*	1	2	3	4	5	6	7	8	9	10	11	12	13
1	2.0	2.8	3.2	3.4	3.1	3.1	3.2	4.0	3.6	3.2	3.8	4.6	3.6	3.4	3.2
2	2.0	3.1	3.6	3.6	4.0	4.0	3.8	2.0	4.6	4.0	3.6	4.2	3.4	3.2	3.2
3	1.5	3.7	3.2	2.8	3.4	2.8	4.2	†	....	....	....	....	....	....	....
4	2.0	2.6	3.4	3.2	3.8	3.8	3.8	4.0	3.4	3.2	3.6	3.6	3.2	3.2	3.0
5	2.0	2.0	3.2	3.4	3.8	4.2	4.2	5.2	4.6	4.2	4.0	3.8	3.6	3.6	3.4

*Pre-inoculation temperatures.
†Died of anemia.

Sow 746 (same litter as 743 and 744) farrowed her first litter on September 1, 1929, with 12 in the litter (fourth generation). Seven of the pigs were inoculated each with 2 cc of hog cholera virus when 11 days old. On the day of inoculation the temperatures varied from 102 to 103°F. Six of the seven showed an elevation of temperature on the second day, ranging from 103.9 to 104.7°F. On the sixth day, the temperatures varied from 103.4 to 105.6° F. The pig showing the highest temperature at this time had a temperature of 102° F. on the day of inoculation and 104.4° F. the second day after inoculation. On the ninth day, the temperature varied from 102.2 to 103.7° F. All the pigs appeared normal during this time.

One pig was reinoculated with 5 cc of virus 135 days after the first inoculation. The results of reinoculation are given in Table IX.

Sow 746 farrowed her second litter on March 5, 1930, with 11 in the litter (fourth generation). Seven pigs were inoculated each with 1.5 to 2.0 cc of hog cholera virus when 15 days old. These pigs did not show any appreciable elevation of temperature following the inoculation of the virus.

From each of the following litters, one pig was reinoculated with 5 cc of hog cholera virus: second litter, sow 741; first litter, sows 742, 744, 746. The time elapsing between inoculation and re-inoculation was 135 days. The results of the reinoculation are given in table IX.

TABLE IX—*Results of reinoculation.*

PIG	SOW	VIRUS (CC)	PRE- AND POST-INOCULATION TEMPERATURES										
			*	1	2	3	4	5	6	7	8	9	10
1	741	5.0	2.2	3.7	3.6	4.4	3.1	4.0	3.4	3.8	4.2	4.0	3.0
2†	742	5.0	2.2	4.6	5.4	4.9	6.6	5.4	6.0	5.8	6.8	7.1	7.0
3	744	5.0	2.3	4.0	4.7	3.4	4.3	4.4	3.4	3.4	3.6	3.3	2.5
4	746	5.0	2.4	4.7	4.7	3.8	4.7	4.2	4.3	4.2	5.5	4.5	4.2

*Pre-inoculation temperatures.
†Died on the 10th day. Postmortem examination showed hog cholera lesions.

TABLE X—*Summary.*

TABLE	SOW	PIGS IN EACH LITTER	IN-OCU-LATED	AGE (DAYS)	ALIVE AT 10TH DAY	ALIVE AT 15TH DAY	REIN-OCU-LATED	TIME BE-TWEEN INOCU-LATIONS (DAYS)	ALIVE 15 DAYS LATER
I-II	A	11	7	16	7	7	4	105	4
	B	12	8	15	8	8	4	109	4
III-IV	B	9	7	18	5	3	1	143	1
	741	6	4	10	4	2	.....	.....	.....
V-IX	741	8	3	5	3	3	1	135	1
IX	742	11	8	14	7	7	1	135	*
VI	742	14	5	16	5	5	.....	.....	.....
	743	7	5	12	5	5	1	141	1
	743	10	3	14	3	3	.....	.....	.....
VII-IX	744	8	6	9	6	6	1	135	1
VIII	744	7	5	15	4	4	.....	.....	.....
IX	746	12	7	11	7	6	1	135	1
	746	11	7	15	7	7	.....	.....	.....
Totals		126	75	.....	71	66	14	.....	13

*Cholera.

None of the pigs that died within 15 days following the first inoculation with hog cholera virus showed lesions of hog cholera on postmortem examination.

SUMMARY

1. The results of these experiments indicate that it is possible to produce permanent immunity against hog cholera in the offspring from immune sows if the baby pigs are inoculated with 1.5 to 2.0 cc of active hog cholera virus before they are 18 days old.

2. Sows immunized with virus only will transmit immunity to their offspring in their second and third litters as well as in their first litter.

3. This immunity is strong enough and permanent enough to withstand successfully a second inoculation of 5 cc of virus 94 to 143 days after the first inoculation.

4. This immunity was permanent enough to permit four sows, 18 months old, to be hyperimmunized by the injection intravenously of 5 cc of virus to the pound of body weight.

Extensive field tests are necessary to draw final conclusions.

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Raising Ostriches for Food

Since the Moscow Zoölogical Park, Moscow, U. S. S. R., has discovered that ostrich meat has a delicious flavor, it has begun the breeding of ostriches on a large scale, according to *Science*. By means of incubators and also by a method that is described as forced mating, each pair is expected to yield between 15 and 16 chicks a year. When fully grown, the birds weigh between 100 and 135 pounds.

It is planned to breed large numbers of the birds in the southern steppes of the U. S. S. R. The thick layer of fat, which formerly served to protect the ostrich against the semi-tropical sun, will serve also as a protection against the cold of the northern latitudes. In its new home the ostrich holds to its native calendar and begins to lay eggs in the fall, which corresponds to the spring of the southern hemisphere.

Peace is always beautiful.—WALT WHITMAN.

THE ETIOLOGY OF PERIODIC OPHTHALMIA*

By L. A. MERILLAT, *Chicago, Ill.*

Since time out of mind, the etiology of periodic ophthalmia of the soliped has been a fascinating study, a study that has yielded a group of theories well known to the readers of classical literature. But, in the absence of satisfactory confirmation, none of the theories has been acceptable. In fact, no one could deny consistently that the etiology of the disease has not been determined.

It was quite natural that the "decades of bacteriology," though which the world of medical science has just passed, should bring the infectious theory into prominence, but the notion that nothing except microparasites is worth considering as etiological is now giving way to a more far-reaching research that is in many instances placing the parasite in a subordinate rôle. This is particularly true of periodic ophthalmia, the etiology of which has baffled all investigators except those who entertain the mesological theory—the theory of environment—which includes a combination of influences (diet, housing, soil, worm parasites) capable of affecting the structure of the eye. The theory includes avitaminosis and anaphylaxis.

The effect of avitamosis A upon the structure of the eye is too well known to require further elucidation, and the effect of anaphylaxis upon the iris, retina, choroid and optic nerve was ably demonstrated by von Scilly, a German oculist, twenty years ago, when he showed that if sheep serum is injected into the vitreous humor of a rabbit and a dose of the same serum is injected into a vein six weeks later, a pronounced iritis develops in the treated eye. Three years ago, Manninger[1] taking up the work of von Scilly, obtained the same results on rabbits with horse serum. This author was able to produce an "anaphylactic ophthalmia" in rabbits that consisted of a fibrinous irido-cyclitis which, during the entire evolution of the disease, was associated with leucocytic infiltration of the choroid, and inflammatory changes in the retina and optic nerve. Bacteriological studies of these eyes gave negative results. The affected structures were sterile.

It seems strange that no reference ever is made to these demonstrations by investigators of the disease in this country. On the other hand, these demonstrations on anaphylactic phenomena

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evolving in the eye and, on the other, the effect of vitamin-A deficiency on the ocular structure of rats, rabbits, chickens, etc., lead one away from rather than toward the infectious theory.

The mesological theory does not exclude worm parasites as a cause of periodic ophthalmia because helminths produce antibodies, and allergic reactions can be employed in the diagnosis of certain parasitisms (warbles, ascarids, etc.). Since anaphylaxis and avitaminoses do gravely affect the eyes, it is not unreasonable to theorize that worm parasites in the intestines possibly are subordinate to conditions favorable to the intervention of various etiological factors anaphylactic in character, for where anaphylaxis is concerned, the provocative agent seldom is lacking.

Our observations on periodic ophthalmia, extending over a long period of years, have brought us nearer and nearer to the conviction that more careful studies, such as were carried out by von Scilly (1914), by Stock (1929) and by Manninger (1930) in Europe, and by Bonaza and E. Merillat (1922-23) in this country, would bear more fruitful results on the etiology of periodic ophthalmia than have been obtained from the bacteriological field.

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Scientists Honored

The King of England has approved the awarding of the Royal Medal by the Royal Society to Mr. P. P. Laidlaw, F. R. S., for his work on diseases due to viruses, including that on the cause and prevention of distemper in dogs, says a recent announcement. The Society also honored Prof. Theobald Smith, of Princeton, N. J., for his original research and observation on diseases of animals and man, by awarding him the Copley Medal.

A Solvent of Remarkable Power

A compound with a wider range of solvent power than any other known substance has been discovered recently by Prof. O. F. Stafford, of the department of chemistry at the University of Oregon, according to *Science*. Professor Stafford's discovery is regarded by his colleagues as an outstanding contribution to the science of chemistry, both in its pure and applied aspects, the article continues. The compound is made from acetic acid and ammonia and is known as acetamide. The ability of acetamide to dissolve many things, at present nearly or quite insoluble, is expected to lead to important industrial applications.

A REVIEW OF HISTORIC VETERINARY BOOKS*

By EMLEN WOOD,† *Philadelphia, Pa.*

Books on veterinary medicine date back to the earliest writings of antiquity, and it is difficult to say just what book is the oldest. It has been customary to start with the ancient Greeks, but much earlier writings have been discovered through archaeological explorations and, as these become more extensive, the "firsts" of all kinds are pushed further and further back, and the first veterinary writings are probably picture-writings of the ancient civilizations, dating from thousands of years before the Christian era. The veterinary art is as old as the domestication of animals and it is known this took place during the primitive stages of man. The earliest writings were in the form of pictures and hieroglyphics. These have been found in many parts of the world, including the near-by states, where a comparatively high state of civilization existed among races ancestral to the modern Indian. Several Egyptian papyri mention medical subjects, and antedate the so-called first mention of medical and surgical subjects, to such an extent that it seems foolish to claim anything as the very first.

In the following remarks I will refer only to the outstanding works of Europe, depending largely on Sir Frederick Smith's "Early History of Veterinary Literature."

Printing was invented in the fifteenth century, about the year 1456, and the first printed veterinary book is, therefore, the first book following that date which discusses diseases of animals. Most of the earliest printing consisted of editions of the writings of classical scholars, which had been written out in longhand, in what are now called medieval manuscripts. In many cases, these had been written during the early centuries of the Christian era and copied over and over again by scribes, or professional writers. The scribes often added their own comments and observations to the original work, so there are great differences between the various manuscript editions of the ancient authors. At about the same period as the introduction of printing, a revival of interest in the writings of the ancients occurred, and the new invention made it possible to issue these old works in greater numbers. Formerly they were locked up in the libraries of the wealthy and of the monasteries, and not available to the general public.

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The books printed during the last half of the fifteenth century are called *Incunabula*. This is a Latin word meaning cradle, and these first books are the cradle, or infancy, of printing. Latin was the universal language of literature and learning and nearly all these early books came out in that language and later were translated into the spoken languages of the different European countries. The author's name was latinized and, as the song says, he might have been "a Frenchman, or a Proossian, or a Roossian, or perhaps I-tal-i-an"; but his name appears on the title-page in a Latin form. There seem to be very few *Incunabula* written by veterinarians, or exclusively on veterinary subjects, but several, and perhaps many, of them take up diseases of animals, and it would be an interesting subject to list all those that might be classified as veterinary *Incunabula*.

First Book on Veterinary Medicine

One of the earliest works, and perhaps the first to deal entirely with veterinary medicine, is by the Roman author, Publius Vegetius Renatus, born 450 A. D. His "Artis Veterinariae," or "The Veterinary Art," thus was written in the fifth century, during the time of the later Roman Empire, but its first appearance as a printed book was not until a thousand years later, when it was published in the city of Basel, in the year 1528. Not much is known about Vegetius, but he was probably an officer in charge of the horses in the army of the Roman Emperor Valentinian. He quoted largely from the writings of the ancient Greek veterinarians and was anxious to raise the standard of veterinary practice of his day to that of the ancient Greeks, among whom were many skilled veterinarians. These men's names are known through the quotations of later writers, but their writings have been almost entirely lost. Vegetius' book is largely a compilation of these earlier works but he was familiar with the epizoötics following the numerous campaigns in which the Roman armies were engaged and gives very modern instructions for their control.

Vegetius is perhaps the first to compare the two professions of animal and human medicine—a comparison that has been made by prominent men in every century down to the present. He points out that the veterinary profession can save the state from loss and is justified economically and asks why should not the curing of animals be considered just as honorable as curing slaves, for mules often cost more than slaves.

His book is a landmark of veterinary literature and shows there was real scientific knowledge in those days 1,500 years ago.

During the succeeding centuries—the Dark Ages—much of it was lost, overcome by superstition and ignorance, and it was not revived till the end of the Middle Ages, when the invention of printing spurred on the revival of learning, and scientific principles again predominated.

Among the first of the printed books is the great encyclopedia of scientific knowledge, written by Bartholomaeus de Glanvilla, Anglicus, or Bartholomew Glanville, the Englishman. He was a Franciscan monk and Professor of Theology in Paris, but he was born in England, in the year 1200 A. D. He was educated in England and living there when he wrote the "De Proprietatibus Rerum," or "The Properties of Things." It was written for readers who had little or no knowledge of science, especially for monks and priests preparing sermons and lectures, and treats of natural history, giving definitions and descriptions of every conceivable subject in nature, from God to the lowest element, the earth. The eighteenth book of this great encyclopedia is on animals, and describes all known animals, including the ape, camel, dog, horse and cow, wild animals and many mythological creatures, as centaurs, unicorns and sea-serpents, for there was a great deal of superstition, and scientific knowledge had not advanced sufficiently to dispel the belief in such beings.

Rabies Described by Early Writer

There is very little on diseases of animals, with the exception of rabies. This term is used in the text, and the name hydrophobia, which later became the name for the mad-dog disease, is not used. Sir Frederick Smith considers Bartholomew's description of the appearance, movements and behavior of a rabid dog nearly perfect. It goes:

> Other hounds flee and avoid the wood hound ("wood" is an old English word meaning mad) as pestilence and venom, and he is always exiled, as it were an outlaw, and goeth alone wagging and rolling as a drunken beast, and runneth yawning and his tongue hangeth out, and his mouth drivelleth and foameth, and his eyes be overturned and reared, and his ears lie backward, and his tail is wrinkled by the legs and thighs; and though his eyes be open yet he stumbleth and spurneth against everything, and barketh at his own shadow.

Bartholomew, of course, did not rely entirely on his own knowledge in compiling an encyclopedia and his description of rabies is quoted from much earlier medical writers, Constantinus Africanus, Pliny and Avicenna. The first printed edition of this work came out about 1470, 250 years after it was written.

About 1500, was published another of the early English works on veterinary medicine and animal husbandry—Sir Walter de

Henley's thirteenth-century manuscript on "Hosbondrie." Henley was a farm bailiff, or manager, of experience and practical knowledge. He discusses the management of horses, cows and oxen, sheep and pigs. Animal hygiene is well described. Nearly 200 years after it was written, it was printed by Wynkyn de Worde, who learned the trade of printer under Caxton, the first printer of England.

Albertus Magnus, or Albert the Great, Bishop of Ratisbon, was another compiler of encyclopedias and a prolific writer during the thirteenth century. One of his works, "De Animalibus," is an encyclopedia on animals. In this he refers to the contagious nature of glanders and farcy and systematically considers the question of infection. In this book and his "Diseases of Birds," he relied largely on Aristotle, and he must be classed as a compiler of the existing knowledge and without practical experience with his subject. Many editions of his works were issued as *Incunabula,* during the fifteenth century.

Founder of Modern Agronomics

Still another of the *Incunabula,* important from the veterinary point of view, is the "Ruralium Commodorum," or "Treatisè on Rural Affairs," of Petrus de Crescentiis, or Pietro Crescenzi, an Italian physician and philosopher, who lived from 1230 to 1307. Hence it dates only a few years later than Bartholomew. He is the founder of modern agronomics, "the first since the Romans to point out the high value of agricultural science. His principles are simple, founded upon experience and free from many prejudices which continued to prevail in Europe for centuries after." (Encyclopedia Americana.) His chapters on diseases of animals were copied from the Greek writers and from Jordanus Ruffus, and his book was of great value in spreading the veterinary principles of Ruffus and marked an epoch in the science of agriculture.

Jordanus Ruffus, an Italian, born at the beginning of the thirteenth century, was Imperial Veterinarian to Frederick II of Sicily, a great patron of science. At the Emperor's command he wrote, about 1250, a text-book on equine medicine, to which the name "Hippiatria" was given. This is a compound word from the Greek hippos (horse) and iatria (practice of medicine). This book has been confused with another of the same name, though sometimes called "Hippiatrika." In fact, there is a third famous veterinary book of the same name, and the confusion is worse confounded by the names of the authors, all beginning with R, being likewise confused.

The original "Hippiatrika" was written during the tenth century and was a compilation of the Greek writings, but chiefly of Apsyrtus and Hierocles. Its compiler is unknown but it was published at the command of Constantine VII; at the same time the "Geoponica," the great agricultural treatise, appeared, and these two words preserved the veterinary and agricultural writings of the Later Roman, or Byzantine, Empire.

Ruffus' book was based largely on this Greek "Hippiatrika," already 200 years old. But he was a practitioner and added many original observations from his own experience. So his book is one of the first by a "graduate veterinarian." The book was very successful. Thirty-five editions came out in Latin, Italian, Sicilian, German, French and Hebrew, before the invention of printing, and the first printed edition in 1492. Other printings followed during the next 150 years and form the basis of several other writers' works. His book was of such importance and exerted so much influence that Smith considers Ruffus the regenerator of the veterinary art and the leading veterinary light during the early stages of the making of modern Europe.

Farcy Described About 1310

Fifty or sixty years later than Jordanus Ruffus, another practicing veterinarian, Laurentius Rusius, flourished in Rome, and wrote "The Book of Marshalry (or Farriery)." In this book he quotes very largely from Ruffus, but includes a great deal based on his own practical experience, and describes farcy and its contagiousness, lameness, and shoeing very capably. This was first written about 1310, brought out in manuscripts as "The Book of Marshalry" many times during the next century and a half, and printed in 1486 and 1490. The next edition was published in Paris, in 1531, and to this unfortunately was given the name "Hippiatria," the same as the book by Ruffus. Hence the confusion of the titles and the authors, as both names have several different spellings. Rusius, of course, is the latinized form of his name; being an Italian, he was probably Laurentio Rusio. When a French edition appeared, he becomes Laurence Rusé, while Ruffus' name has nine or ten different spellings. So the two names get badly mixed, especially in the later editions bearing the same title.

The third of these "R" authors is Johannes Ruellius (the Latin form of his real name), Jean Ruelle, a Parisian physician who compiled a volume of the diseases of horses and their cures in 1530, about three centuries later than Ruffus and more than 200 years later than Rusius. Ruellius was born in 1479 at Soissons,

and became a prominent physician and botanist, a great writer on the works of the ancients, and famous for his "De Natura Stirpium," or "The Nature of Plants." He was appointed physician to the King, Francis I, and by him commanded to edit the tenth century Hippiatrika, or "Two Books of Veterinary Medicine interpreted by Johannes Ruellius of Soissons," as the subtitle reads. This is the old original Hippiatrika again and this time, its editor not being a veterinarian, added nothing new, though Ruellius obviously meant his great book to be useful in aiding the development of the veterinary art.

Ruini's Anatomy of the Horse

Still another veterinary writer has a name starting with R, but this man belongs to the sixteenth century. The most famous of the medieval veterinary authors, Carlo Ruini's name will live in history for all time as the author of the first anatomy of the horse, "Anatomia del Cavallo; Infermita et Suoi Rimedii," Venice, 1598. Ruini was of a prominent family, wealthy and well educated, a lawyer and a senator of Bologna, Italy. He was interested in anatomy and realized the need for a text-book on the anatomy of the horse, of which there was absolutely none in existence. His associates—probably they were the same type as our polo and hunting set of today—were interested in horses. At any rate, the Italians were skilled horsemen, far ahead of the English of the time, and were skilled in veterinary medicine. Their country had been the embarkation point of the Crusaders on their way to and from Palestine for nearly 200 years and this must have given them thorough experience in all the animal plagues so intimately associated with armies and shipping.

Ruini wrote in Italian, not Latin, probably because his book was meant for farriers and practical horsemen, who of course, used Italian as their every-day language. He undoubtedly did a lot of dissection himself, but probably also had several assistants. His book is well illustrated, several of the cuts showing the ventral aspect of the horse lying squarely on its back with the legs flexed, and the interior of the abdomen exposed. Others show the muscles of different parts of the body, without names, however—an indication of the lack of knowledge of comparative anatomy. Ruini's book first appeared in 1598 and is noteworthy in the history of medicine because of his views on the circulation of the blood. He wrote 30 years before Harvey published his great discovery, and many times since 1628 (when Harvey's book came out), different persons have tried to rob Harvey of his fame by asserting that he copied his ideas from others.

Several scientists had a partial view of the truth concerning the circulation and a fairly good conception of the lesser circulation —from the heart through the lungs. Ruini was one of these, but anatomy and physiology were so little known in his day that no one knew the course of the blood in animals or man, and most people still believed the ancient idea that the arteries contained air only.

From patriotic and professional zeal, Ercolani, dean of the Veterinary School at Bologna, had a tablet erected to commemorate Ruini's discovery of the circulation of the blood. This started quite a controversy in the medical journals in 1876, and Gamgee, of Scotland, reviewed the whole matter in the British *Lancet*, his conclusion being that it goes too far to credit Ruini with the discovery. Harvey's great discovery has for three centuries attracted the attention of medical historians and while some have included Ruini among the so-called precursors of Harvey, there seems to be a tendency among them to ignore him. The veterinary profession should keep Ruini's name in this list, as there is no question that he wrote of the passage of the blood between the heart and the lungs about the same year that Harvey began his medical studies.

Ruini's anatomy was translated into German by Uffenbach in 1603, into French by Francini in 1607 and again by Jourdain in 1647, and it is striking to note that later editions in Italian, in 1618 and in 1706, were issued apparently without any change or revision. It would seem that either there was not enough interest in learning equine anatomy—but if so, why publish the book at all?—or else, no one felt qualified to improve on Ruini's descriptions. His book has not been translated into English but was used largely in preparing the first anatomy in the English language, "The Anatomy of an Horse," 1683, by Andrew Snape Jr. Snape was Serjeant-farrier to King Charles II, "Being a Son of that Family that hath had the honor to serve the Crown of the Kingdom in the Quality of Farriers for these two Hundred Years." So he was a practicing veterinarian, for that is the meaning of farrier in those days, before the founding of veterinary schools, and it meant more than horseshoer or groom as in our army today. His family in the next generation made an abrupt change of occupation, for Snape's son, Andrew, became Headmaster of Eton and later Provost of Kings' College, Cambridge.

Another writer, advanced as a rival of William Harvey, is the Spanish veterinarian, Francisco de la Reyna, whose "Book of

Veterinary" was printed first in 1522, and went through many editions, published in different cities of Spain. He was probably "one of two most expert physicians kept in pay by Alphonso, King of Arragon (i. e. old Spain), and commanded to put forth a most excellent treatise on the several diseases of beasts." This is quoted from a life of the next author to be mentioned, but the original Latin, instead of beasts, is "*pro equis et canibus,*" which means horses and dogs, and this is the oldest Spanish book, and one of the oldest in Europe, on diseases of dogs, that has been found.

De la Reyna's knowledge of the circulation of the blood was mentioned by Lebret, in 1825, in a French translation of a Spanish bibliography of veterinary medicine and should be investigated further, as the early medical history of Spain has not received as much attention as that of the other countries.

Henry Cornelius Agrippa von Nettesheim, a German physician, wrote "The Vanity of Science and Art," first printed in 1530. In this, "veterinary surgery is discussed briefly and kindly, as a useful art, too proudly scorned by the physicians."

Later in the same century, in 1564, John Philip Ingrassia, of Sicily, published in Venice a work entitled, "Whether veterinary medicine should be formally one and the same with the nobler medicine of man; the difference in materials does not lessen its dignity nor nobility."

Book on Breeds of Dogs

One of the earliest descriptions of the different breeds of dogs was written in 1570, by John Keys, best known by his latinized name, Caius. He was an English physician, born in 1510, educated at Cambridge and in Italy; edited the works of Hippocrates and Galen and contributed so largely to his alma mater, Gonville Hall, one of the colleges of Cambridge University, that it has since been known as Gonville and Caius College. While studying in Europe, he formed a friendship with Conrad Gesner, one of the best known naturalists of the time, and at his request wrote "De Canibus Brittanicus," or "On English Dogs." Fortunately for the modern reader, this was translated from the original Latin, a few years later, by Abraham Fleming. Good descriptions are given of many of our present breeds: the bloodhound, the greyhound, and the setter, with his Latin name, Index, because he points to the game, are easily recognized. Dr. Caius evidently had many women patients, and perhaps specialized in nervous diseases of women. At any rate he did not have a very

good opinion of women-owners of lap dogs, as the following quotation shows:

> Of the delicate, neat and pretty kind of dogs called the Spaniel gentle, or the comforter. Called by Latin authors Melitaeus, from the Island of Malta, where this kind of dogs had their principal beginning. These dogs are little, pretty, proper and fine and sought for to satisfy the delicateness of dainty dames, and wanton womens' wills; instruments of folly for them to play and dally withall, to trifle away the treasure of time, to withdraw their minds from more commendable exercises, and to content their corrupted concupiscences with vain disport. (A silly shift to shun irksome idleness.) These puppies the smaller they be, the more pleasure they provoke, as more meet playfellows for mincing mistresses to bear in their bosoms—to keep company withal in their chambers, to succour with sleep in bed, and nourish with meat at board, to play in their laps and lick their lips as they ride in their wagons—and good reason it should be so, for coarseness with fineness hath no fellowship, but featness with neatness hath neighborhood enough. That plausible proverb, verified upon a Tyrant that he loved his sow better than his son, may well be applied to these kind of people who delight more in dogs that are deprived of all possibility of reason, than they do in children, that be capable of wisdom and judgment. But this abuse peradventure reigneth where there hath been long lack of issue, or else where barrenness is the best blossom of beauty.

Caius' description of dogs was included by Gesner in his "Historia Animalium," 1555 a great natural history, in four volumes of viviparous and oviparous quadrupeds, birds and fishes. Conrad Gesner, born 1516, was a Swiss physician and Professor of Philosophy and Natural History; described as the greatest naturalist since Aristotle, he discovered the true principles of botanical arrangement. He describes many monsters and impossible animals, but is careful to point out that these are only supposed to exist, from the writings of others.

Two Manuals of Zoölogy

Fifty years later, Edward Topsell, an English minister, compiled two elaborate manuals of zoölogy, drawn chiefly from Gesner's works, but in addition took up diseases and their cures, copying from Blundeville and Gervase Markham. Topsell is the man who first told us "to tie that bull outside." In describing the bull he says:

> The strength of the head and necke of a bul is very great, and his forehead seemeth to be made for fight: having horns short, but strong and piked, uppon which he can tosse into the aire very great and weighty beasts which he receiveth againe as they fall downe, doubling their elevation with renewed strength and rage, untill they be utterly confounded. Their strength in all parts of their body is great, and they use to strike backward with their heeles; yet it is reported by Coelius Titormus, a Neat-heard of Aetolia (that is, a cowboy) that being in the field among the cattell, (he) took one of the most fierce and strongest buls in the heard

by the hinder leg, and there in despight of the bull striving to the contrary, held him with one hand, untill another bull came by him, whome he likewise tooke in his other hande, and so perforce held them both.

This story is illustrated by a picture of a bull—not in as good flesh as the well-known Bull Durham—and a remarkably good drawing of a grazing cow, both animals' tails vigorously switching flies. The horse and mule also are illustrated and, it is interesting to note, modern-looking shoes with the rows of nails clearly shown on the walls of the hoof.

Thomas Blundeville, a Norfolkshire landowner, from 1560 to 1566, produced a work on horsemanship,—"The fower chiefest offices belonging to Horsemanshippe, * * * of the Breeder, of the Rider, of the Keeper and of the Ferrer." It is one of the most important veterinary writings of England and about the earliest based on real scientific observation. Blundeville, not possessing much practical knowledge of diseases, relied on Martin, the Court Veterinarian, and by observing his practice and drawing on Martin's wide experience, produced a really worth while book, which formed the basis of all subsequent works for years to come. This is especially so in the case of the prolific writer and plagiarist, Gervase Markham, who wrote many books on horsemanship, veterinary medicine, and numerous other subjects, amounting to 36 separate works, at the end of the sixteenth and beginning of the seventeenth centuries.

It would take too long to discuss this writer and he makes a good place to stop, for according to Smith, Gervase Markham's writings stopped all progress in veterinary medicine until the long series of wars in Europe, with their resultant animal plagues, forced the foundation of veterinary schools and the modern period of the profession had its birth.

Preparedness

For more than thirty years, Mr. Duncan Neale, of Sherstone Magna, Wiltshire, England, lived with his brother, Moses, and neither of them married. On the death of his brother, eleven years ago, however, he made the most minute arrangements for his own burial, including the erection of a tombstone recording family deaths, and with an inscription (to be completed) relating to his own, and married almost immediately. Mr. Neale, a veterinary practitioner, died November 2, 1933.—*Veterinary Record.*

12th International Veterinary Congress
New York—August 13-18, 1934

PRIMARY MYXOSARCOMA OF CHICKENS*

By CARL OLSON, JR.,† *Rochester, Minn.*
Division of Experimental Medicine, The Mayo Clinic

A review of the literature reveals that myxoblastoma is relatively uncommon in man[1] and in lower animals.[2] Eber and Malke,[3] in a study of 239 neoplasms of chickens, observed one myxoma of the skeleton and eight myxosarcomas. Malke[4] described four myxosarcomas (table I). Tyzzer and Ordway,[5], and John[6] presented one case each of myxosarcoma. In Klee's[7] series of 89 tumors of chickens, myxosarcoma occurred once. Makower[8] found two myxosarcomas in 54 tumors in birds killed for food. I observed a chicken with myxosarcoma of the ovary and peritoneal implantations. A description follows:

A White Leghorn hen, aged twenty months, in a fair state of nutrition, was noted to be very anemic. Examination of the blood revealed hemoglobin approximately 0.5 gm per 100 cc of

TABLE I—*Myxoblastomas of domestic fowl reported in the literature.*

REPORTED BY	TYPE	PRIMARY SITE	METASTASIS TO
Eber and Malke	Myxoma mixed cell	Skeleton	
Malke	Sarcoma myxomatodes	Ovary	Heart muscle, kidney, ceca
Malke	Mixed cell sarcoma, myxomatodes	Ovary	Implants to peritoneum
Malke	Mixed cell myxosarcoma	Ovary	Implants to peritoneum
Malke	Myxosarcoma cavernosum	Ovary	Spleen, mesentery of oviduct
Tyzzer and Ordway	Myxosarcoma	Subcutis of thigh	
John	Myxosarcoma	Breast muscle	Lung, liver, spleen

*Received for publication, October 17, 1933.
†Fellow in Comparative Pathology, The Mayo Foundation, University of Minnesota, Rochester, Minn.

blood (normal, 9.0 gm), with leukopenia and immature cells of the erythroblastic series. The condition was diagnosed erythroleukosis.

At necropsy, in addition to confirmation of the diagnosis, erythroleukosis, the nonproducing ovary was found to be involved with a pale, flesh-colored mass 3 by 2 by 2.5 cm. There also were several irregular nodular masses scattered along the mesentery near its intestinal attachment. These neoplastic masses varied in size from 1 to 2 cm and were covered with slimy gelatinous material. A few minute implantations were observed on the mesentery between its attached border and root. The other visceral organs did not show gross evidence of involvement.

Histologically the neoplastic tissue from the mesentery revealed stellate and spindle-shaped cells with a few cells in mitosis. Many of the cell processes were anastomosing with each other, forming a loose network. In areas near blood vessels, the number of cells was appreciably greater (fig. 1). Mucin was demonstrated by staining the tissue with Mayer's mucicarmine method. There was relatively little mucin, yet it was definitely present, particularly in the cell processes and adjacent to them. Other tissues examined histologically included the liver, spleen, lung and heart, none of which revealed metastatis.

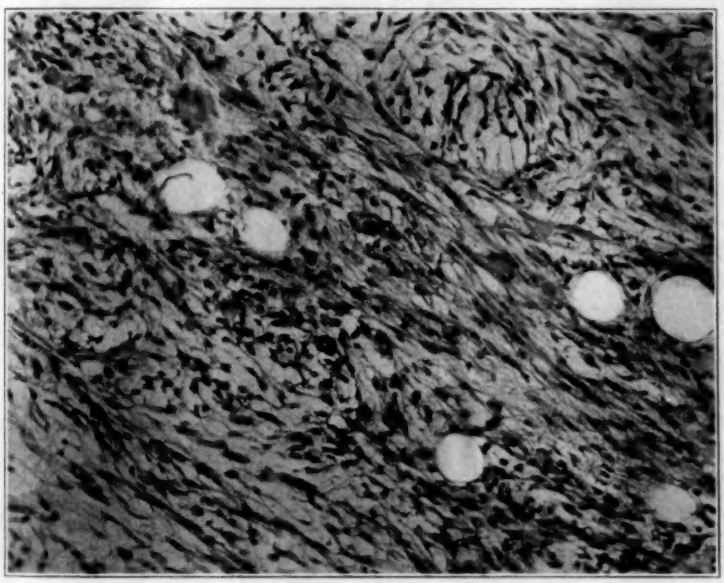

FIG. 1. Myxosarcoma of mesentery; the greater number of cells may be noted in the region of blood-vessels (x150).

Comment

Primary myxoblastomas may be divided into myxomas and myxosarcomas, the latter being characterized by properties of implantation and metastasis and hence malignant. It is not uncommon to see myxomatous tissue in certain adult connective tissue tumors, such as fibromas and chondromas; the tumors then are termed myxofibromas or myxochondromas. The prefix "myxo" is applied to describe the myxomatous tissue. Another type of tumor containing cells with mucous proclivities is recognized as myxomatous degeneration of more adult connective tissue cells, and such a tumor sometimes is designated "myxomatodes," for example, chondroma myxomatodes. This is not to be confused with secondary myxomas, the cells of which arise by metaplasia or imperfect growth of mesoblastic cells.

According to the orthodox view, the primary mxyoma may be assumed to arise from early undifferentiated connective tissue, with a mucous propensity which is generally distributed in the embryo, or from embryonal mucous tissue whose distribution is less widespread. Thus the elements of primary myxomas are to be considered embryonal. It is Ewing's belief that primary myxoma does not tend to differentiate into lipoma or fibroma. The secondary myxoma must then arise from imperfect growth, or metaplasia, of neoplasms of mesoblastic origin, forming areas of mucous tissue in these tumors.

The ovary of the chicken appears to be the site of predilection for the origin of myxoblastoma, as four of the seven cases recorded in the literature give this as the primary site. The tumor in the case presented here likewise has its origin in the ovary. The implantations on the mesentery indicate the malignancy of the growth.

Erythroleukosis is to be regarded as the cause of death of the chicken, and bears no relationship to the myxoblastoma other than as a coincidental finding.

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[3]Eber, A., and Malke, E.: Geschwulstbildungen beim Hausgeflügel. Zusammenfassender Bericht über 392 bei 16,460 Geflugelsektionen ermittelte Neubildungen. Zeit. f. Krebsforsch., xxxvi (1932), pp. 178-192.

[4]Malke, E.: Geschwulstbildungen beim Haushuhn. Zeit. f. Krebsforsch., xxxi (1930), pp. 47-66.

[5]Tyzzer, E. E., and Ordway, T.: Tumors in the common fowl. Jour. Med. Res., xxi (Oct., 1909), pp. 459-477.

[6]John: Quoted by Joest, E., and Ernesti, S.: Untersuchungen über spontane Geschwülste bei Vögeln. Zeit. f. Krebsforsch., xv (1916), pp. 1-76.

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GRANULOMA OF THE EYE OF A DOG: REPORT OF A CASE*

By CARL F. SCHLOTTHAUER, *Rochester, Minn.*
Division of Experimental Medicine, The Mayo Clinic

The following case is being reported because of the peculiar pathologic changes and their probable etiology.

October 3, 1933, a male collie, six years of age, was brought to me to be destroyed because of a large tumor in the orbit, which the owner thought to be cancerous in nature. This tumor was pedunculated and protruded from the orbit (fig. 1). It had a flattened top 5 cm in diameter. The mass was freely movable and appeared to involve the eye only. This appeared to be completely obliterated.

The owner stated that in the autumn of the previous year the dog had been shot accidentally with a shotgun. At this time it had been noted that his eye was injured. It was thought that a pellet of shot might have entered the eye, although the

FIG. 1. Tumor in situ.

*Received for publication, November 6, 1933.

eye did not appear to be ruptured. The dog had suffered no other ill effects. The tumor had been observed first about two months later and had progressed in size slowly.

The dog was killed with ether, and postmortem examination was performed. The tumor was easily dissected from its attachment in the orbit. The eye itself was completely obliterated. There was no evidence of metastasis. Several small-sized lead shot were found in the subcutaneous tissues, lungs and liver, and one was found in the wall of the gall-bladder.

Histologic study of the tumor revealed that it was composed of inflammatory tissue, that it was a granuloma. It could have been removed safely and easily and the dog's usefulness prolonged.

A SPINDLE-CELL SARCOMA INVOLVING THE SPINAL CORD*

By J. F. BULLARD, *LaFayette, Indiana*
Department of Veterinary Science
Purdue University Agricultural Experiment Station

This is a case report of a spindle-cell sarcoma which involved the spinal cord of a small pig weighing about 20 pounds.

When this pig was brought to the laboratory for examination, it was unable to stand. There was no history of injury and external trauma could not be found. It would lie quietly on either side but, when disturbed, it kicked with all four feet. The visible mucous membranes and bowel action were normal, and its appetite and general condition were good, indicating that it had not been down more than 24 or 48 hours. Actually, it was stricken approximately 24 hours previous to the examination.

The pig was destroyed and the autopsy revealed a tumor in the anterior cervical portion of the spinal cord, in the region of the atlas and axis. The tumor appeared as a grayish white, lobulated mass resembling adipose tissue. It was 63 mm long, 19 mm wide and 17 mm deep. It nearly filled the vertebral canal.

Microscopic sections, taken at various levels, showed that practically the entire cord was involved in the anterior portion, while at the posterior border only about the dorsal half of the cord was infiltrated. In all sections the neoplasm had infiltrated the meninges and cord substance.

*Received for publication, November 27, 1933.

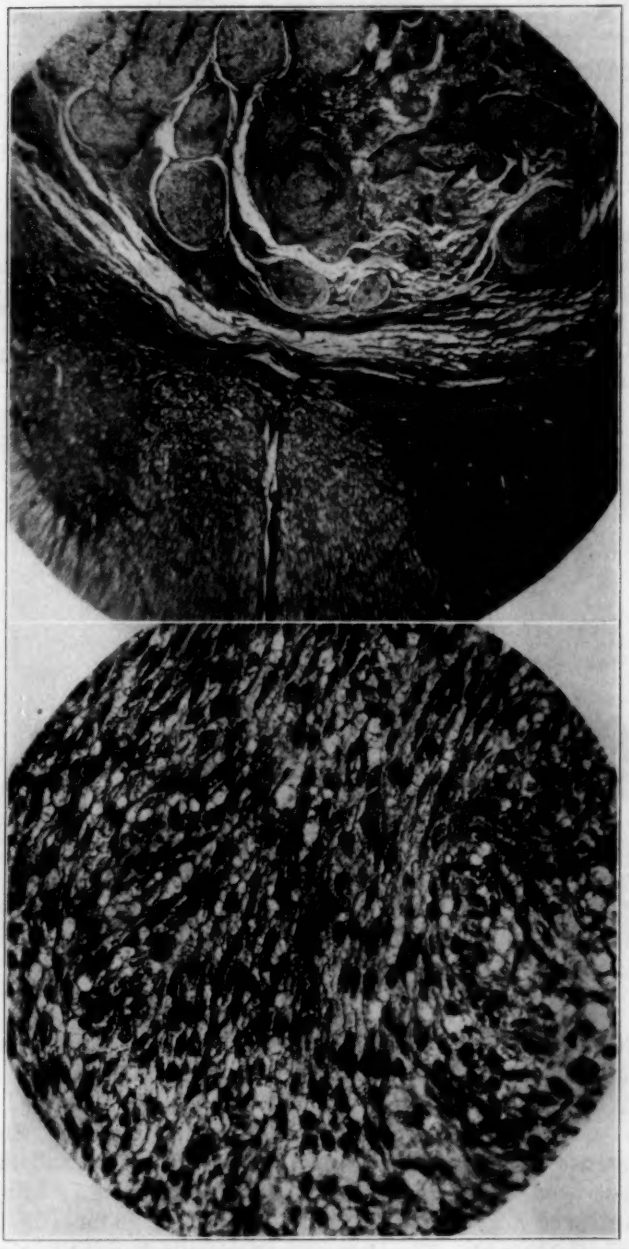

FIG. 1 (above). This section shows the general relationship of the tumor to the cord. The ventral median fissure and horns are easily recognizable.

FIG. 2 (below). Section of a cell-nest showing the characteristic spindle cells. Considerable lipoidosis also is present.

A LARGE OSTEOSARCOMA OF THE MANDIBLE*

By FRANK THORP, JR., *and* ROBERT GRAHAM
Laboratory of Animal Pathology and Hygiene
University of Illinois, Urbana, Illinois

Specimen: The head of a black male colt, 10 months of age, bearing a large growth on the anterior surface of the mandible, was submitted to the Diagnostic Laboratories of the Division of Animal Pathology and Hygiene by Dr. A. E. Campbell, Rantoul, Illinois, on April 11, 1933. According to the history accompanying the specimen, the growth first became noticeable when the colt was 6½ months of age. It appeared as a small

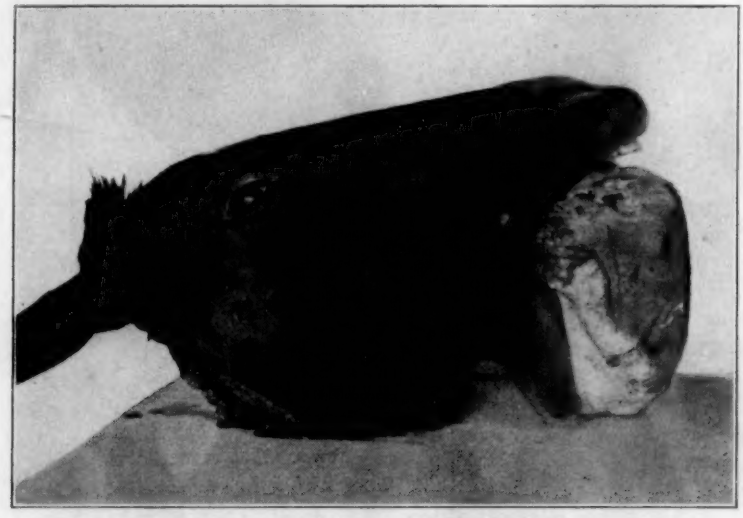

FIG. 1. Side view of osteosarcoma of the mandible. The white ridge is epithelium encroaching upon the tumor from all sides.

enlargement on the gums just below the lower incisor teeth. During the suceeding 3½ months, the growth increased in size rapidly and prominently protruded from the mouth. The lower lip was pushed downward by the increasing pressure. Surgical interference at this time was not deemed advisable and the colt was destroyed. The growth on removal weighed 8.76 kilograms and measured approximately 24 cm x 13 cm x 28 cm (fig. 1).

Gross Pathology: The tumor apparently had its origin on the mental surface of the body of the mandible, with growth taking place ventrally, laterally and anteriorly. Development of

*Received for publication, November 27, 1933.

the tumor was so rapid that the anterior surface of the neoplasm was not covered with epithelium. The epithelium was visible as a white, superficial ridge of tissue only partially covering the tumor from the sides. On palpation the neoplasm was very firm, while on the anterior surface, bone spicules could be removed with forceps. A cross-section of the tumor revealed osseous tissue containing numerous areas of liquefaction necrosis, varying from 1.5 to 6 cm.

Histopathology: The cellular structure of the neoplastic tissue was characterized by the presence of spindle-shaped sarcoma cells and an abundance of immature bone, characteristic of an osteosarcoma (fig. 2).

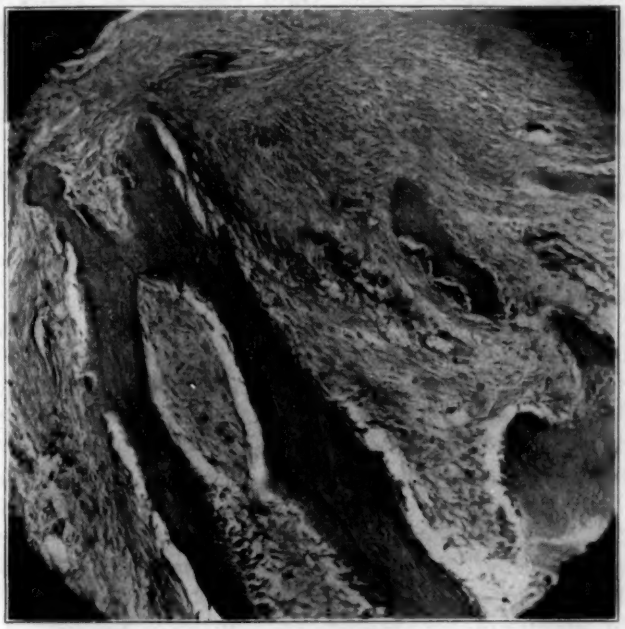

FIG. 2. Photomicrograph showing spindle-shaped cells and immature bone (x80).

RENAL NEOPLASMA OF DOG*

By WILLIAM H. DUNN, *Darien, Conn.*

Subject: King Charles spaniel, eight years old, male.

History: This dog had always been a well-cared-for house pet and had received constant veterinary supervision. One year ago,

*Received for publication, December 9, 1933.

multiple small skin tumors about the head were removed and the dog given a tenifuge. At this time the animal was in apparent good health. About six months ago, the dog developed a slight perineal hernia which was readily reducible and caused no apparent discomfort. All this time the dog was on a raw meat and vegetable diet.

Symptoms: On November 23, the dog was presented exhibiting spasmodic pain in the abdomen, which had had a sudden onset that morning. The dog ate, urinated and defecated normally that morning. On direct palpation of the abdomen, extreme pain and rigidity of the abdominal wall was evidenced. Slight pressure caused painful urination.

Anatomical diagnosis: The right kidney was found to be enormously enlarged, indurated and painful. The left kidney apparently was unchanged. Fluoroscopic study was negative for urolithiasis. Therefore, a tentative diagnosis of malignant neoplasma of the right kidney was made with a recommendation of destruction.

Pathological anatomical diagnosis: The abdominal cavity contained about 300 cc of serous exudate. The intestines apparently were normal, but the liver was swollen and indurated. The spleen was enlarged and indurated.

Kidneys: The left kidney apparently was normal. It weighed 30 gm and measured 5 x 3 x 2 cm. The right kidney was enormously enlarged, weighed 200 gm, measured 8 x 7 x 6 cm, was indurated and dark purplish-red in color. Cut section exhibited marked interstitial hemorrhage with almost complete obliteration of the pelvis.

Histopathological diagnosis: The section of the normal kidney shows simple congestion. Section of the other kidney shows an enormous number of dilated capillaries surrounding masses of short spindle cells. Hyperchromatism is marked in places. The normal structure of this kidney is entirely destroyed.

Diagnosis: Angiosarcoma.

Remarks: This case of renal dysfunction is interesting because of the lack of clinical symptoms until late in the involvement of the kidney.

The symptomatology of this case almost parallels that of the case of bilateral renal calculi recently reported by Schlotthauer.[1]

Acknowledgment: The histopathological diagnosis was made by Dr. C. F. Murray.

REFERENCE

[1]Schlotthauer, C. F.: Spontaneous bilateral renal calculi in a dog. Jour. A. V. M. A., lxxxiii (1933), n. s. 36 (5), pp. 694-696.

MANUAL OF VETERINARY BACTERIOLOGY. Major Raymond A. Kelser, V. C., U. S. A., Officer in Charge, Veterinary Laboratory Division, U. S. Army School. 2nd edition. 552 pages, 93 figures. Williams and Wilkins, Baltimore, 1933. Cloth, $5.50.

No better evidence of the value of this book could be asked than the necessity for a second edition so soon after the first. In the interval between the two editions, much has been added to our knowledge of veterinary bacteriology. This is especially true concerning variations in cell morphology, and in the colony characteristics of various species of bacteria under different conditions. The author has included a chapter in this edition, dealing with present concepts of the more important phases of bacterial variation, by Maj. James S. Simmons, Medical Corps, U. S. A.

The plan, adopted in the first edition, of adhering to the classification of bacteria, as developed by Bergey and his associates, has been continued consistently, although the author is not in complete accord with the system. He has adopted it rather than contribute further to the present muddle of bacteriological nomenclature.

The introductory chapter to the Section on the Protozoa has been prepared by Colonel Charles F. Craig, of Tulane University, an eminent authority on the subject.

The author is to be commended for the thoroughness with which the second edition has been prepared. From the standpoint of physical qualities, the publishers have done their part. The book is fully up to the Williams and Wilkins standard.

VETERINARY MATERIA MEDICA AND THERAPEUTICS. E. Wallis Hoare. Edited and revised by J. Russell Greig, Director, the Moredun Research Institute, Edinburgh, and Hon. Research Professor in Animal Pathology in the Royal (Dick) Veterinary College, Edinburgh. 5th edition. 510 pages. Alexander Eger, Chicago, Ill. Cloth, $5.50.

This is the fifth edition of the work originally by Hoare, published in 1895.

Part I has been condensed materially by Professor Greig, who has striven also for greater lucidity. Part II, covering materia medica and biological products, has been largely rewritten, with a view to more clearly recognizing the claims of pharmacology, the link in the teaching curriculum between physiology and therapeutics.

Part III deals with therapeutics and is divided into eight chapters, each dealing with a separate system or organ of the body. Each chapter is subdivided, with sections devoted to (A) the horse, (B) cattle, (C) the dog, and so forth.

There are three appendices to the work. The first contains numerous formulae which are to be considered as merely representative. The second consists of a table of thermometric equivalents (Centigrade and Fahrenheit), and a table of standard wire gauges (inches and millimeters) to be used in describing sizes of cannulae and hypodermic needles. Appendix III deals with the British law relating to dangerous drugs.

The recent publication of a new edition of the British Pharmacopoeia made necessary numerous alterations in nomenclature and formula.

Dr. Hollingworth in New Post

As a result of the reorganization of the city government of Utica, N. Y., the Department of Food Hygiene, formerly a part of the Bureau of Health, has been added to the Bureau of Food Sanitation, which will henceforth be known as the Bureau of Food Hygiene and Sanitation. Dr. W. G. Hollingworth will be in charge of the new Bureau. Other cities please copy.

What Is a Dogie?

How many persons, outside of those who live on or near the Western ranges, know the meaning of the word "dogie," except in a vague way?

Taking exception to the definition given by *Time* that a dogie was a yearling, a western cattleman describes a dogie as follows: "Dogie—pronounced doe'-gie—a range term of many years' standing, has suffered a rebirth on account of one of the most popular songs of the day. In short, a dogie is a motherless calf. It is often used to denote an animal stunted in growth, or having out-of-proportion curves. It is also the other man's cattle."

This description, which appeared in *The Cattleman*, carried with it a picture of a dogie, 1933 model.

LYMPHOMATOSIS, MYELOMATOSIS AND ENDOTHELIOMA OF CHICKENS CAUSED BY A FILTRABLE AGENT. I. Transmission experiments. J. Furth. Jour. Exp. Med., lviii (1933), 3, p. 253.

A new transmissible strain of leukosis of chickens is described that causes (a) lymphomatosis with or without tumor formation, and with or without leukemia, (b) myelocytomatosis with or without leukemia, and (c) endothelioma. All of these diseases are transmissible by material free from viable cells, and the available evidence indicates that they are caused by a single filtrable agent.

STUDIES ON AN UNCOMPLICATED CORYZA OF THE DOMESTIC FOWL. I. The isolation of a bacillus which produces a nasal discharge. John B. Nelson. Jour. Exp. Med., lviii (1933), 3, p. 289.

By a method combining filtration and cultivation, an unidentified Gram-negative bacillus was isolated from the nasal exudate of a fowl experimentally infected with an uncomplicated coryza. Isolation was accomplished by cultivation on scaled blood-agar plates after unsuccessful attempts to produce colonies on open plates. Injection of the organism into the palatine cleft of normal birds was followed regularly by an inflammation of the nasal mucosa and a discharge from the nares. A parainfluenza bacillus, which was also recovered from the nasal tract of the infected fowl, was innocuous. Cultural characters of the bacillus are discussed.

STUDIES ON AN UNCOMPLICATED CORYZA OF THE FOWL. II. The relation of the "bacillary" coryza to that produced by exudate. John B. Nelson. Jour. Exp. Med., lviii (1933), 3, p. 297.

Three types of an uncomplicated fowl coryza differing in the onset and duration of symptoms, developed after the intranasal injection into normal birds of exudate from natural cases. Protection tests were carried out with two of the types in an attempt to explain why the "bacillary" disease regularly ran a shorter

course than the "exudate" disease. Reciprocal protection was demonstrated in one case, but in the other the birds which had recovered from the "bacillary" disease were susceptible to reinfection with exudate. The coryza produced by exudate and bacilli, respectively, can be transmitted from infected birds to normal ones by direct contact.

RELATION OF THE ADRENAL CORTEX TO VITAMINS A, B AND C. Julia E. Lockwood and Frank A. Hartman. Endocrin., xvii (1933), 5, p. 501.

Tests were made to determine the influence of cortical extract of vitamin A, B and C, respectively, using an average of 7 to 11 animals for each curve. Guinea pigs were used in the vitamin C experiments and rats in the vitamin B and A. When administered by mouth the cortical extract afforded no protection against avitaminosis C and B. However, when the extract was injected intraperitoneally, it (a) improved the growth curve and scurvy score in avitaminosis C, (b) improved the growth curve and delayed the onset of symptoms in avitaminosis B, and (c) had no influence in ameliorating the symptoms in avitaminosis A. Adrenal weights showed hypertrophy of the adrenals in vitamin C and B deficiencies and atrophy in vitamin A deficiency. The injection of cortical extract containing cortin delayed the onset of symptoms in avitaminosis C and B but had no influence in avitaminosis A. Either cortin or some unidentified substance must be responsible for this effect. The authors conclude that cortin or some unidentified substance aids in the utilization of vitamins C and B. They suggest that an ample supply of these vitamins may be advantageous in adrenal cortical insufficiency.

THE DETERMINATION OF THE BITTER SUBSTANCE IN BITTERWEED MILK. Norabelle D. Weathers. Jour. Dairy Sci., xvi (1933), 4, p. 401.

The presence of the bitter substance imparted to milk by the bitterweed (*Helenium tenuifolium*) can be detected by the characteristic orange-red color that develops when contaminated milk is treated directly with solutions of picric acid and sodium hydroxid, equal parts (0.2 cc) of a 1.2 per cent solution of picric acid and a 10 per cent solution of sodium hydroxid being added to 2 cc of milk. The sample is shaken thoroughly and it is suggested that it be compared with a control of normal milk treated

similarly. One part of the substance can be detected in 50,000 of unaltered milk. The quantitative determination of the bitterweed principle may be accurately made on the alcohol-ether extracts of centrifuged milk by the use of standards of purified bitter crystals, or by the use of either creatinine or picramic acid standards.

NEW SNAIL AND RABBIT HOSTS FOR FASCIOLA HEPATICA LINN. Wendell H. Krull. Jour. Parasitol., xx (1933), 1, p. 49.

The snail *Fossaria modicella* Say has been determined experimentally to be a new secondary host of *Fasciola hepatica* Linn, and the cottontail rabbit, *Sylvilagus floridanus mallurus* (Thomas) to be a new primary host. Cercariae escaped from these snails which were subjected to infection when fully grown, in 32 days, but laboratory-raised snails infected when still small died before the cercariae-producing stage was reached. Laboratory-raised snails of the species *Succinea avara* Say and *Lymnaea* (Galba) *palustris* Müll, and an opossum *Didelphys virginiana* Kerr could not be infected.

TWO AVIAN TUBERCLE BACILLUS DISSOCIANTS AND TWO HUMAN TUBERCLE BACILLUS STRAINS OF DIFFERENT VIRULENCE. Florence B. Seibert, Esmond R. Long and Nelle Morley. Jour. Inf. Dis., liii (1933), 2, p. 175.

A typical avian tubercle bacillus (Van Es 1921) grew in rough (R type), dry colonies on Long's synthetic medium and in smooth (S type), greasy colonies on Petroff's egg medium. The S bacilli were longer, more slender, more beaded and less acid-fast than the R bacilli and tended to stratify, whereas the R type piled up in irregular clumps. The S bacilli proved more virulent for hens than the R bacilli. Differences were conspicuous in the spleen. Soon after inoculation with S bacilli numerous minute tubercles formed, which were not caseous and loaded with bacilli. In hens infected with the R type at the same time, tubercles were fewer, larger and caseous with few visible bacilli. At a later period the lesions were more nearly alike, but more numerous in the hen infected with the S type. Chemically the S bacilli contained less water and more fatty material than the R bacilli. Two strains of tubercle bacilli of human type grown on the same medium also showed the same chemical differences.

Regular Army

No changes during the month of November.

Veterinary Reserve Corps

New Acceptances

Nolan, James Dixon—2nd Lt.....Raymond, Wash.

The Budapest Prize

The Eighth International Veterinary Congress was held in Budapest, Hungary, in 1905. At the close of the Congress, it was found that the Organizing Committee had an unexpended balance in the treasury. The funds were used to create a permanent foundation, and it was provided that the income should be used for defraying the cost of awarding a medal, to be known as the *Budapest Prize*, at each Congress, to the author or authors of some important veterinary work that shall have been published during the previous ten years.

It was provided further that the foundation should be administered by the Royal Hungarian Veterinary Medical Association (Magyard Orszagos Allatorvos Egyesulet), or by its successors, and that the medal should be prepared and forwarded to the proper committee before the opening of each Congress.

This year, the Committee will consist of the members of the Permanent Committee of the International Veterinary Congresses, the honorary presidents, the president of the Congress, the presidents of the sections of the Congress, to be selected at New York, and a delegate from the Royal Hungarian Veterinary Medical Association. This Committee will meet, at the latest, on the day before the closing session of the Congress, the president of the Congress presiding, for the purpose of selecting the author or authors to receive the award. The winner of the medal will be announced at the closing session of the Congress.

ORGANIZATION OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION, 1933-1934

(Concluded from page 832, December issue)

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Idaho ..... E. M. Gildow, University of Idaho, Moscow.
Illinois ..... H. C. Rinehart, Dept. of Agr., Springfield.
Indiana ..... R. C. Julien, Pitman-Moore Co., Indianapolis.
Iowa ..... J. A. Barger, 311 U. S. Court House, Des Moines.
Kansas ..... C. W. Bower, 1128 Kansas Ave., Topeka.
Kentucky ..... E. A. Caslick, Paris.
Louisiana ..... Hamlet Moore, 2941 Grand Route St. John, New Orleans.
Maine ..... M. E. Maddocks, 7 School St., Augusta.
Maryland ..... M. F. Welsh, College Park.
Massachusetts ..... H. W. Jakeman, 44 Bromfield St., Boston.
Michigan ..... E. K. Sales, 535 Forest St., East Lansing.
Minnesota ..... R. A. Merrill, Clara City.
Mississippi ..... W. L. Gates, Box 417, Clarksdale.
Missouri ..... Ashe Lockhart, 800 Woodsweather Rd., Kansas City.
Montana ..... Hadleigh Marsh, Agr. Exp. Station, Bozeman.
Nebraska ..... Frank Breed, Norden Laboratories, Lincoln.
Nevada ..... L. R. Vawter, University of Nevada, Reno.
New Hampshire.... R. W. Smith, 161 Court St., Laconia.
New Jersey..... E. R. Cushing, 947 Prospect Ave., Plainfield.
New Mexico..... T. I. Means, Box 1174, Santa Fe.
New York..... J. G. Wills, Box 751, Albany.
North Carolina..... J. H. Brown, Tarboro.
North Dakota..... Lee M. Roderick, State College Station, Fargo.
Ohio ..... B. H. Edgington, 215 S. Remington Rd., Columbus.
Oklahoma ..... C. H. Fauks, 1719 S. W. 15th St., Oklahoma City.
Oregon ..... W. B. Coon, Forest Grove.
Pennsylvania ..... B. Scott Fritz, Dept. of Agr., Harrisburg.
Rhode Island..... T. E. Robinson, 92 High St., Westerly.
South Carolina..... M. R. Blackstock, 157 W. Hampton Ave., Spartanburg.
South Dakota..... G. E. Melody, Hoven.
Tennessee ..... F. W. Morgan, 1025-27 McCallie Ave., Chattanooga.
Texas ..... T. O. Booth, Temple.
Utah ..... E. A. Bundy, 1656 25th St., Ogden.
Vermont ..... G. N. Welch, 43 Union St., Northfield.
Virginia ..... I. D. Wilson, Virginia Poly. Inst., Blacksburg.
Washington ..... C. E. Sawyer, Western Washington Exp. Sta., Puyallup.
West Virginia..... H. M. Newton, Box 1721, Charleston.
Wisconsin ..... J. S. Healy, 330 Federal Bldg., Madison.
Wyoming ..... H. D. Port, 304 State Capitol Bldg., Cheyenne.

RESIDENT TERRITORIAL SECRETARIES

Alaska .....J. B. Loftus, Juneau.
Canal Zone.....Lt. Col. A. L. Mason, Corozal.
Hawaii .....Col. R. C. Musser, Headquarters Hawaiian Dept., Honolulu.
Philippine Islands...Victor Buencamino, 1026 Felix Huertas, Manila.
Puerto Rico.....H. L. Van Volkenberg, Agr. Exp. Sta., Mayaguez.
Virgin Islands.....Guy A. Roberts, Christiansted, Saint Croix.

RESIDENT PROVINCIAL SECRETARIES

Alberta .....J. C. Hargrave, Box 673, Medicine Hat.
British Columbia...J. G. Jervis, Milner.
Manitoba .....Alfred Savage, Manitoba Agr. College, Winnipeg.
New Brunswick.....David J. McLellan, Swift Canadian Co., Ltd., Moncton.
Nova Scotia.....William W. Jakeman, 3 Doyle St., Halifax.
Ontario .....R. A. McIntosh, Ont. Vet. College, Guelph.
Prince Edw. Island.W. H. Pethick, Charlottetown.
Quebec .....A. A. Etienne, 1225 Drummond St., Montreal.
Saskatchewan .....Mark Barker, Post Office Bldg., Regina.

FOREIGN CORRESPONDING SECRETARIES

Bermuda .....C. J. Cooper, "Kelton," Pembroke.
China .....H. C. Evangelista, Box 1170, Shanghai.
Colombia .....Roberto P. Guerrero, Apartado 149, Bogota.
Cuba .....L. A. Beltran, Box 883, Havana.
England .....R. W. Tuck, c/o American Consulate, 18 Cavendish Sq., London, W. 1.
Hungary .....A. Kotlan, Royal Hungarian Vet. College, Budapest, VII.
Jamaica .....Stephen Lockett, Dept. of Agr., Hope, Kingston.
Mexico .....L. Santa Maria, Apartado Postal 2067, Mexico, D. F.
New Zealand.....W. C. Ring, 62 Albert St., Auckland.
Peru .....J. F. Mitchell, c/o Hacienda Pachacayo, Pachacayo.
St. Kitts.....C. F Jardine, Box 18, Basseterre.
Scotland .....A. W. Whitehouse, Glasgow Veterinary College, 83 Buccleuch St., Glasgow.

Who's the Goat?

A young man who had never been off the city pavements was appointed to a job appraising farm live stock. On his first assignment he couldn't identify a billy goat, so after carefully noting its characteristics telephoned his office in town: "It has whiskers on its chin and a sad look in its eyes, but I dunno what it is," he screamed into the transmitter. "Oh, don't you know what that is?" said his boss. "That's the farmer."

Chicago *Daily News.*

12th International Veterinary Congress
New York—August 13-18, 1934

TWELFTH INTERNATIONAL VETERINARY CONGRESS

Waldorf-Astoria Hotel, New York, N. Y.
August 13-18, 1934

OFFICERS

Chairman of the Organizing Committee: Dr. A. Eichhorn.

Vice-Chairman: Dr. L. A. Merillat.

Treasurer: Dr. John R. Mohler.

General Secretary (to whom all communications should be addressed): Dr. H. Preston Hoskins, 221 N. La Salle St., Chicago, Ill.

Program

The Congress will consist of general and sectional meetings. Preliminary plans provide for four general meetings, scheduled for Monday, Tuesday, Thursday and Saturday mornings. In addition there will be six half-days devoted to sectional meetings. The subjects to be presented at the general meetings were selected by the Permanent Committee, at the meeting in Paris, on May 20, 1933. The reporters also were selected at that time. These subjects and reporters are:

New Plans for the Combating of Enzoötic Diseases Under a State Veterinary Service. Professor Dr. E. Leclainche, Director of the Bureau of Epizoötics, Paris, France.

Relationship of Veterinary Science to Animal Breeding and Public Health. Legal Protection of the Practice of Veterinary Science. Dr. John R. Mohler, Chief of Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

Veterinary Control of the Marketing of Milk. Dr. R. von Ostertag, Ministerial Director (Reserve), Tübingen, Germany.

New Researches on Filtrable Viruses. Dr. F. Gerlach, Director of the Federal Institute for Combating of Animal Diseases, Mödling near Vienna, Austria, and Professor Dr. R. Manninger, Royal Hungarian Veterinary College, Budapest, Hungary.

New Researches on Contagious Abortion (Bang's Disease). Professor Oluf Bang, Royal Veterinary and Agricultural College, Copenhagen, Denmark; Dr. W. E. Cotton, Superintendent of Experiment Station, Bureau of Animal Industry, U. S. Department of Agriculture, Bethesda, Md., and Professor G. Finzi, Director of the Royal Superior Institute of Veterinary Medicine, Milan, Italy.

The following subjects will be presented at the sectional meetings:

SECTION I—Pathology, Bacteriology and Contagious Diseases:
Tuberculosis.
Foot-and-Mouth Disease.
Hog Cholera.

Anthrax.
Gas Edema Diseases.
Classification of Paratyphoid Diseases.
Infectious Anemia of Horses.
Lymphadenitis of Sheep.
Equine Encephalomyelitis (Mosquitoes as Vectors).

SECTION II—Medicine, Surgery and Obstetrics:
Parturient Paralysis.
Sterility.
Diseases of Young Animals.
Infectious Mastitis.
Recent Progress in Veterinary Surgery.

SECTION III—Veterinary Parasitology and Parasitic Diseases:
Therapeutics of Worm Diseases.
Immunity Against Parasites.
Coccidiosis.

SECTION IV—Fowl Diseases:
Pullorum Disease.
Fowl Plague.
Coryza.
Psittacosis.
Fowl-Pox.
Leukemia.
Neurolymphomatosis Gallinarum.

SECTION V—Tropical Diseases:
The Piroplasmoses.
Spirochetosis.
African Horse Sickness.
Anaplasmosis.
Cattle Plague.

SECTION VI—Hygiene of Meat and Milk:
Pasteurization of Milk.
Meat Inspection.

SECTION VII—Animal Breeding and Dietetics:
Genetics.
Deficiency Diseases.
Scientific Principles of Feeding.
Blood-Group Question.

Almost one hundred reporters have been invited to discuss the subjects to be presented at the sectional meetings. These reporters are the world's outstanding authorities on the subjects listed. They will come from all corners of the world to bring the latest developments in their respective fields.

At the annual meeting of the National Association of Bureau of Animal Industry Veterinarians, held in Chicago, December 6-7-8, 1933, the organization made a contribution of $500.00 to the Twelfth International Veterinary Congress, as a memorial to Dr. D. E. Salmon, Dr. A. D. Melvin and others, on the occasion of the 50th anniversary of the establishment of the Bureau of Animal Industry in the United States Department of Agriculture.

The New Food and Drug Bill

President Roosevelt has ordered a new deal for consumers. At his direction, Assistant Secretary of Agriculture Tugwell, with the assistance of officials of the Food and Drug Administration, has drafted an entirely new food and drug law (Senate bill 1944) which was introduced by Senator Copeland, of New York, at the short session of Congress. The bill has had public hearings before the Senate Commerce Subcommittee since December 5.

The new legislation is designed to furnish a more effective weapon against abuses in the food and drug industries than is now provided by Dr. Wiley's rather antiquated statute of 1906, enacted during the administration of President Theodore Roosevelt. Modern commercial practices, which Dr. Wiley and his associates could not have anticipated, call for new methods of control. Court decisions have revealed hundreds of defects in the old law and these will have to be corrected if consumers of foods and drugs are to have adequate protection.

Back in 1906, when the original law was drafted, manufacturers depended to a great extent upon their labels to sell goods. Truthful labels seemed to be sufficient protection for consumers. For that reason, there was no provision for bringing other forms of advertising under control. As a result, manufacturers today can go as far as they wish in making preposterous claims in their printed advertising and radio broadcasting. All that it is necessary for manufacturers to do is to see to it that their labels conform with the law. No matter how worthless a product may be or how false the claims for it, an innocuous label will keep the manufacturer out of trouble.

Other problems, in connection with the law, have arisen through changed modes of living. There was a time when most foods were prepared in the housewife's own kitchen. Today, a large part of the various foods consumed are produced outside the home. Thanks to modern scientific methods, many of these foods are better and cheaper than the housewife could prepare. In the production of others, however, there are grave abuses which

can not be controlled under a law that makes no provision for legal standards for foods (except for butter and certain canned goods), or for the federal supervision of food industries.

Cosmetics, which were of practically inconsequential importance at the time the 1906 law was passed, are today a major industry intimately affecting the health and pocketbooks of millions of consumers. Yet, unless the label or cosmetic container bears some medicinal claim for conditions recognized as disease, even though the cosmetic be poisonous, it does not come within the law. Likewise many dangerously potent drugs, such as the radium charged water responsible for the death of a prominent Pittsburgh citizen a short time ago, enjoy an unrestricted sale under labels giving absolutely no hint of their dangerous character, simply because they are not adulterated and their labels—as far as these go—are not untruthful. Except for a few specifically named narcotic and habit-forming drugs, precautionary labels can not be required under the law.

The Tugwell Bill retains all of the valuable features of the old law revitalized to meet modern conditions and it also makes special provision for future contingencies. First of all, it brings cosmetics and curative devices of every description—from hair dyes to sun-lamps and orthopedic shoes—under government control. It prohibits all false and misleading advertising of foods, drugs and cosmetics through any medium whatever. It requires that labels not only be truthful but definitely informative, so that the consumer may know what he is buying, whether or not it possibly can harm him and just how he may use it with safety. Any other information necessary to protect his health or his purse can be required at the discretion of the Secretary of Agriculture.

The bill gives the federal government authority to set up standards of quality and identity for all food products and to establish safe tolerances for poisons in foods. If any food is subject to contamination in an insanitary factory, the government is empowered to put the manufacturer under a permit that will guarantee sanitary conditions and a wholesome product. To put a stop to false claims for medicinal drugs, it will be sufficient for the government to show in court that a drug is worthless for the purpose for which it is advertised without having to prove also, as required by the present law, that the manufacturer knows that his product is of no value. More drastic penalties for violations, with injunctions against chronic offenders, many of whom have regarded the payment of fines rather lightly, insure more faithful

observance of the law and correspondingly greater protection for consumers.

It is expected that there will be a bitter fight in Congress when the Tugwell bill is being debated. Strange as it may seem, some manufacturers already have indicated their opposition to the new bill, because of fear that it would put them out of business. Manufacturers of meritorious products should have absolutely nothing to fear as far as the new legislation is concerned. Radio listeners certainly will heave a big sigh of relief if and when the new law is passed, as it will then be no longer necessary to listen to ballyhoo which now is broadcast, all day long and well into the night, by manufacturers, particularly of cosmetics, who apparently must depend upon exaggerated claims for the sale of their products.

DR. A. E. CAMERON

New Honor for Doctor Cameron

The Professional Institute of the Civil Service of Canada recently chose a new president, elevating Dr. A. E. Cameron, of Ottawa, from the first vice-presidency to the presidency. News of Dr. Cameron's appointment will be received with great interest by members of the A. V. M. A., since he is a member of the Executive Board, having been elected to that office, June 23, 1932.

A life of adventure, professional activity and service is evidence enough of Dr. Cameron's fitness for this new honor.

TRI-COUNTY VETERINARY ASSOCIATION

A meeting of the Tri-County Veterinary Association was held at Winona, Minn., November 7, 1933. Featured speakers at the meeting were: Dr. C. P. Fitch, president of the American Veterinary Medical Association; Dr. A. C. Spannaus, of Waconia, president of the Minnesota State Veterinary Medical Society, and Dr. C. F. Schlotthauer, of the Mayo Foundation, Rochester, Minn.

Officers for the coming year were elected as follows: President, Dr. V. C. Willis, Waucoma, Iowa; vice-president, Dr. N. A. Roettiger, Winona, Minn.; secretary-treasurer, Dr. P. H. Riede, Mabel, Minn.

P. H. RIEDE, *Secretary.*

SOUTHERN STATES VETERINARY MEDICAL ASSOCIATION

The eighteenth annual meeting of the Southern States Veterinary Medical Association was held at the Ansley Hotel, Atlanta, Ga., November 23-24, 1933. Dr. W. A. Hornaday, of Greensboro, N. C., president of the Association, presided. The invocation was given by Rev. Louie D. Newton, of Atlanta. The address of welcome was delivered by Hon. J. L. Key, mayor of Atlanta, and the response was made by Dr. W. A. Barnette, mayor of Greenwood, S. C.

The featured speaker at the opening session was Dr. C. P. Fitch, president of the American Veterinary Medical Association, whose topic was "Our National Association." He stressed the need for the coöperation of the states in the work of the A. V. M. A. The session was closed with a paper by Dr. E. L. Shuford, Jr., of Asheville, N. C., on "My Experience With Sequietan in the Treatment of Canine Distemper."

At the afternoon session of the first day, Dr. H. B. Treman, of Rockwell City, Iowa, presented a paper on "Large-Animal Practice," in which he emphasized new and better ways of performing the more common operations. He brought with him a

prepared specimen of the foot of a horse for use in the demonstration of many diseases of the foot and their treatment. Dr. C. A. Cary, of the Alabama Polytechnic Institute, gave an address on "Botulism in Poultry."

A jolly crowd attended the banquet in the evening, at which Dr. F. E. Kitchen, of Greenville, S. C., presided as toastmaster. Dr. D. W. Daniels, professor of English at Clemson College, S. C., entertained the crowd with a humorous lecture, which was enjoyed thoroughly. Several short talks were made, after which the evening was given over to dancing.

Three papers were presented at the opening session of the second day: "Parasites in Small Animals," by Dr. H. C. Nichols, of Ocala, Fla.; "The General Application of Practical Serum Therapy in Veterinary Practice," by Dr. C. E. Salsbery, of Kansas City, Mo., and "The Results of Recent Investigations of Animal Diseases at University Farm," by Dr. C. P. Fitch, who discussed problems in the control of diseases in wild animals, especially the moose, and used motion pictures to illustrate his talk.

In the afternoon, a clinic was held at the hospital of Dr. J. Lee Hopping. Dr. Treman was in charge of the large-animal division, and Drs. L. J. Kepp and J. C. Wright, of Atlanta, conducted the small-animal demonstrations.

The following officers will serve during the coming year: President, Dr. H. C. Nichols, Ocala, Fla.; first vice-president, Dr. R. G. Kitchen, Sumter, S. C.; second vice-president, Dr. J. H. Coffman, Atlanta, Ga.; third vice-president, Dr. John H. Gillmann, Memphis, Tenn.; secretary-treasurer, Dr. M. R. Blackstock, Spartanburg, S. C.

M. R. BLACKSTOCK, *Secretary.*

NEBRASKA STATE VETERINARY MEDICAL ASSOCIATION

The thirty-sixth annual meeting of the Nebraska State Veterinary Medical Association was held at the Lincoln Hotel, Lincoln, December 12-13, 1933, with approximately 125 members and guests in attendance.

The meeting was called to order by Dr. J. E. Weinman, of Lincoln, President. The reading of the minutes of the 1932 meeting was followed by Dr. Weinman's presidential address, in which he summarized the activities of the Association for the past year and made some recommendations for the future. One of the significant statements made by Dr. Weinman was to the

effect that veterinarians should remain loyal to the ethical serum companies that have done so much to further the interests of the practicing veterinarian.

The Association voted to contribute $100 toward the Twelfth International Veterinary Congress; decided to hold a clinic at David City during the summer instead of the usual short course at Lincoln; and adopted a resolution commending the advertising campaign carried on by the Associated Serum Producers, Inc., during the past year. Thirteen applicants were admitted to membership.

Dr. F. B. Young, of Waukee, Iowa, handled the Division of Cattle Practice in a very practical manner. This was followed by a round-table discussion. Dr. A. T. Kinsley, of Kansas City, Mo., conducted the Division of Swine Practice and, as usual, a very lively discussion of swine problems followed. Dr. Frank Breed, of Lincoln, was scheduled to handle the Division of Poultry Practice, but was prevented from doing so by a severe laryngitis. Dr. R. R. Dykstra, of Kansas State College, was present and kindly volunteered to complete the program for the afternoon in lieu of the subject that had been announced for Dr. Breed. Dr. Dykstra presented an illustrated lecture covering the subject of "Everyday Surgery." His lecture was greatly appreciated.

The evening of the first day was given over to the annual banquet, with 136 present. Dr. L. A. Merillat, of Chicago, officiated as toastmaster. Dr. Dykstra, the speaker of the evening, presented an illustrated address entitled "A Century of Progress in Veterinary Medicine." With lantern-slides, he presented some of the prominent men who have distinguished themselves in veterinary medicine. He also showed pictures of some of the modern buildings that are devoted to the study of veterinary science. Dancing followed the banquet.

The program Wednesday morning was opened with an address by Dr. H. L. Feistner, chief of the Nebraska Bureau of Animal Industry, who gave a report of the activities of his office during the year. He was followed by Dr. C. F. Schlotthauer, of the Mayo Foundation, Rochester, Minn., who gave an illustrated lecture covering small-animal practice. This was followed by a round-table discussion that lasted until 11:30.

The election of officers resulted as follows: President, Dr. S. W. Phillips, David City; vice-president, Dr. Fred W. Collins, of Madison; secretary-treasurer, Dr. E. C. Jones, Grand Island (reëlected). Drs. S. S. Gibson, of Randolph, and S. M. Score, of Albion, were elected to the Executive Board to fill the vacan-

cies caused by the retirement of Drs. J. H. Copenhaver and M. Campbell. Dr. S. W. Phillips, President-elect, was selected to represent Nebraska in the A. V. M. A. House of Representatives until January 1, 1935. The same motion provided that Dr. Fred W. Collins, Vice-president-elect, be designated as alternate.

At the Wednesday afternoon session, Dr. E. T. Baker, who is sometimes referred to as the Will Rogers of the veterinary profession, conducted the Division of Sheep Practice. Dr. Baker had traveled a distance of 1,800 miles from Moscow, Idaho, to attend the meeting. He handled his subject in a very practical manner and made many valuable suggestions for handling sheep problems.

Dr. C. P. Fitch, president of the American Veterinary Medical Association, addressed the meeting and pointed out the value of the A. V. M. A. to the individual practitioner in a way that should stimulate a desire on the part of every veterinarian to support the activities of the national association. As an example he related how he, as president of the A. V. M. A., had been advised of a conference in Washington to discuss a proposal to turn over to the farm bureaus the testing of cattle for Bang's disease, in connection with the government's program for curtailing and regulating production during the present emergency. Dr. Fitch related what had been done to block such a move.

Dr. L. A. Merillat handled the Division of Equine Practice and, as evidence of the returning interest in this subject, the discussion was not brought to a close until 5:30 o'clock.

It was decided to meet in Omaha in 1934.

E. C. JONES, *Secretary.*

WESTERN NEW YORK VETERINARY MEDICAL ASSOCIATION

The twentieth annual meeting of the Western New York Veterinary Medical Association was held at Buffalo, December 14, 1933, with 50 members in attendance.

After a short clinical program, the meeting was called to order by the President, Dr. H. V. Baker, and routine business was transacted. The Association voted to contribute $100 toward defraying the expenses of the Twelfth International Veterinary Congress, to be held in New York City, August 13-18, 1934.

Seventy persons sat down to a banquet given in the Hotel Touraine at 6:30, after which the ladies attended the theater and the members listened to a talk by Dean W. A. Hagan, of the

New York State Veterinary College, on "Equine Encephalomyelitis." This was followed by two reels of motion-pictures showing the different phases of the disease. Dr. R. H. Volgenau, of Buffalo, gave a review of Dr. D. H. Udall's book on "The Practice of Veterinary Medicine."

The following officers were elected to serve during the coming year: President, Dr. Bernard P. Wende, Buffalo; vice-president, Dr. W. C. Buck, Dansville; secretary-treasurer, Dr. F. F. Fehr, Buffalo (reëlected).

F. F. FEHR, *Secretary.*

EAST TENNESSEE VETERINARY MEDICAL SOCIETY

A meeting of the East Tennessee Veterinary Medical Society was held at the White Medical Supply Company, Knoxville, December 9, 1933, with 21 veterinarians in attendance. The program was devoted to a discussion of Bang's disease. The laboratory director of the University of Tennessee demonstrated tests for the disease. After the meeting, Dr. White served a Dutch lunch and liquid refreshments.

F. W. MORGAN, *Res. Sec. for Tennessee.*

CHICAGO VETERINARY MEDICAL SOCIETY

At the meeting of the Chicago Veterinary Medical Society, Chicago, Ill., on December 12, 1933, Dr. Joseph E. Schaefer, attending oral surgeon at Cook County Hospital, Chicago, and president of the Chicago Dental Society, gave an illustrated lecture on "Bone and Soft Tissue Lesions." Dr. Schaefer showed about a hundred lantern-slides, some in color, in describing interesting cases that had come to his attention.

Changes in Cuba

Dr. Bernardo J. Crespo has resigned as chief of the Division of Animal Industry of Cuba and has been succeeded by Dr. Eduardo Gómez Echaso. Dr. Rafael de Castro has resigned as director of the Bio-Pathological Laboratories of the Department of Agriculture, and Dr. Rogelio Arenas has been appointed to fill the vacancy.

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NECROLOGY

SAMUEL K. HAZLET

Dr. Samuel K. Hazlet, of Oelwein, Iowa, died at his home November 26, 1933, after an illness of two years following a cerebral hemorrhage.

Born at Highland, Iowa, December 23, 1867, Dr. Hazlet studied veterinary medicine at the Chicago Veterinary College. He was graduated in 1895 with honors, and began his practice at Elgin, Iowa. After a year in Elgin, he removed to Oelwein, which had been his home since 1896. He was made an assistant state veterinarian of Iowa in 1896, an office he held for more than thirty years.

Dr. Hazlet joined the A. V. M. A. in 1911. He was a member and past president of the Iowa Veterinary Medical Association. He was affiliated with the Masonic order, with the local order of the Modern Woodmen of America and with the Rotary Club. He was active in civic work and at the time he was stricken was councilman-at-large for the city of Oelwein.

Surviving Dr. Hazlet are his widow (née Nellie Mettlin), three daughters, two brothers, and one sister.

FRED G. PATCH

Dr. Fred G. Patch, of Roseville, Ill., died at his home, November 30, 1933, after a brief illness.

Born at Kewanee, Ill., November 26, 1871, Dr. Patch had spent practically the whole of his life in Roseville. Following his graduation from high school, he entered the Illinois State Normal University. Later he decided to take up the study of veterinary medicine. He matriculated at the Chicago Veterinary College and received his degree in 1910. Attesting to the high esteem in which Dr. Patch was held, six of his colleagues in the veterinary profession served as pallbearers at the impressive funeral rites.

Dr. Patch joined the A. V. M. A. in 1918. He is survived by his widow (née Bertha M. Boyd), one son and one sister.

HERMAN C. EDEWAARD

Dr. Herman C. Edewaard, of Holland, Mich., died at the Holland Hospital, December 5, 1933, following an emergency operation.

Born in The Netherlands, May 25, 1876, Dr. Edewaard was a graduate of McKillip Veterinary College, class of 1916. He joined the A. V. M. A. in 1922. He is survived by his widow (née Ida Botma), one daughter, two sons, one brother and two sisters.

JOHN VERLIN RAMLER

Dr. J. V. Ramler, of Dodge Center, Minn., died of lobar pneumonia, December 6, 1933, at Detroit Lakes, Minn., where he had gone to assist in the tuberculosis-eradication project in that area.

Born in Rockwell City, Iowa, May 4, 1896, Dr. Ramler was a graduate of the Kansas City Veterinary College, class of 1918. Following his graduation, he offered his services to the Army Veterinary Corps and did duty at Camp Greenleaf, Ga. At the time of his death he was a second lieutenant in the Veterinary Reserve Corps. In 1919, he entered the employe of the Minnesota State Live Stock Sanitary Board as a field veterinarian and continued in that service until his death.

Dr. Ramler joined the A. V. M. A. in 1919. He was also a member of the Minnesota State Veterinary Medical Society. He was a member of Corner Stone Lodge No. 99, A. F. and A. M., of Fergus Falls, Minn., and of Consistory No. 1, of the Saint Paul Scottish Rite bodies. He is survived by his widow (née Marie Williams) and one son. Funeral services were held in Minneapolis, Minn., on December 10, and burial was in Acacia Cemetery at Mendota.

H. C. H. K.

RICHARD THOMAS JAMES

Dr. Richard T. James, of New Westminster, B. C., died suddenly at his home on December 6, 1933.

Born at Carp, Ont., February 24, 1894, Dr. James was graduated from the Ontario Veterinary College in 1915. He served overseas during the World War with the Imperial Veterinary Corps. After the War, he joined the meat-inspection service of the Health of Animals Branch, Canadian Department of Agriculture. For some years he had been in charge of meat inspection at the Swift Canadian abattoir at New Westminster.

Dr. James was a member of King Solomon Lodge No. 17, A. F. and A. M., and of the New Westminster Chapter of Royal Arch Masons. He is survived by his widow and two sons.

J. G. J.

JAMES R. CARSON

Dr. J. R. Carson, of Cicero, Ind., died at his home, December 7, 1933, a victim of typhoid fever. He had been ill for about a month.

Born in Hamilton County, Ind., October 10, 1867, Dr. Carson was educated in the schools of the County prior to his matriculation at Indiana Veterinary College, from which institution he was graduated in 1907. He had been a resident of Cicero during his entire life with the exception of three years which he spent on a farm.

Dr. Carson joined the A. V. M. A. in 1912. He was identified with the Odd Fellows and Red Men, and was active as a member of the Commercial Club of Cicero. At one time he was identified with the Town Board. He is survived by his widow (née Lavina Flannagan), three sisters, one half-sister and one brother.

EDWARD R. ELLERBROCK

Dr. Edward R. Ellerbrock, of Lima, Ohio, died December 9, 1933, of a fractured skull, the result of an automobile accident near Vaughnsville, Ohio. It is believed that the driver of the car in which Dr. Ellerbrock was riding lost control of the car as a result of the bad driving conditions caused by inclement weather. The accident happened just before midnight.

Dr. Ellerbrock was a graduate of the Cincinnati Veterinary College, class of 1915. He is survived by two sons, two daughters, his father, three sisters and three brothers.

RAY SCHUCHERT

Dr. Ray Schuchert, of Keystone, Iowa, died at his home, December 17, 1933.

Born at Marengo, Iowa, November 28, 1887, Dr. Schuchert attended Belle Plaine (Iowa) High School and then entered the McKillip Veterinary College. He located in Keystone, about 1917, after having seen military service in the U. S. punitive expedition into Mexico.

Hill Memorial Library
Louisiana State University

Dr. Schuchert joined the A. V. M. A. in 1924. He is survived by his widow (née Gertrude Hoff), two daughters, one son, his mother, and several brothers and sisters.

GEORGE WESLEY TURLEY

Dr. George W. Turley, of Remington, Ind., died at his home, December 19, 1933, of ether pneumonia, which followed a broken leg two weeks previously.

Born in Hendricks County (Ind.), July 8, 1881, Dr. Turley received his veterinary education at the Indiana Veterinary College. Following his graduation in 1910, he located in Remington, where he enjoyed a large practice.

Dr. Turley is survived by his widow (née Alma Bowman), three sons, four brothers and two sisters.

Our sympathy goes out to Dr. F. J. Baker, Gouverneur, N. Y., in the death of his wife on November 22, 1933, and also to Dr. W. G. Brock, of Dallas, Tex., in the sudden death of his wife recently.

PERSONALS

MARRIAGES

Dr. Raymond Caldwell Snyder (U. P. '33), to Miss Georgina Worrell, October 25, 1933, at Philadelphia, Pa.

Dr. A. E. Bott (Chi. '13), of East Saint Louis, Ill., to Mrs. Ethlyn W. Hopkins, of Saint Louis, Mo., November 3, 1933, at Columbia, Mo.

Dr. Robert B. Helming (K. S. C. '31), of Cresco, Iowa, to Miss Winifred Mitchell, of Cresco, October 6, 1933.

BIRTHS

To Dr. and Mrs. C. C. Middleton, of Birmingham, Ala., a son, November 23, 1933.

To Dr. and Mrs. Louis Leonpacher, of Lafayette, La., a son, Robert Joseph, December 22, 1933.

PERSONALS

Dr. M. W. Scothorn (O. S. U. '33) is practicing at Ashville, Ohio.

Dr. C. L. McGinnis (K. S. C. '33) is practicing at Far Hills, N. J.

Dr. James G. Tufts (U. P. '33) is now located at Morristown, N. J.

Dr. F. B. Jones (K. C. V. C. '11) has removed from Manhattan, Kan., to Enid, Okla.

Dr. G. W. Leahy (McK. '15), formerly of Decatur, Ill., is now located at Princeton, Ill.

Dr. Ray O. Porter (K. C. V. C. '09) has removed from Neosho, Mo., to Yellville, Ark.

Dr. W. T. Hinshaw (T. H. '14), formerly of Holgate, Ohio, has removed to Adrian, Mich.

Dr. M. A. Hollingsworth (Ont. '95), of Geneseo, Ill., underwent an operation in November.

Dr. S. W. Wiest (St. Jos. '19), formerly of Santa Fe, N. M., has located at Alliance, Neb.

Dr. W. J. Cant (Chi. '09), of Erie, Ill., has been appointed quarantine inspector for Whiteside County.

Dr. Graydon McKee (O. S. U. '33), formerly of Colebrook, Ohio, has entered practice at Beaver, Pa.

Dr. O. J. Hummon (O. S. U. '30), formerly of Lewisburg, Ohio, is now practicing at Leipsic, Ohio.

Dr. B. A. Zupp (Iowa '23) has changed locations from Waltham, Minn., to Blooming Prairie, Minn.

Dr. H. A. Bernas (San Fran. '18), formerly of Iloilo, P. I., is now at La Granja, Occidental Negros, P. I.

Dr. James L. Orr (A. P. I. '23) reports a change of address from Long Island City to Poughkeepsie, N. Y.

Dr. J. S. Grove (Ont. '92) reports a change of address from Austin, Texas, to 2534 Wilkinson St., Fort Worth, Texas.

Dr. O. A. Anderson (K. S. C. '33) is a member of the staff of the Raritan Hospital for Animals, Inc., Stelton, N. J.

Dr. E. P. Spaeth (U. P. '98), of Gillette Wyo., is vice-president of the Wyoming State Livestock and Sanitary Board.

Dr. C. J. Buehler (K. C. V. C. '17), of Pekin, Ill., underwent an operation for appendicitis at a Peoria hospital recently.

Dr. E. F. Jarrel (Chi. '08), of Tyler, Tex., recently resigned as a member of the Live Stock Sanitary Commission of Texas.

Dr. Arthur J. Knilans (Chi. '16) has returned to Janesville, Wis., after having been located in Freeport, Ill., for several years.

Dr. E. R. Dimock (K. C. V. C. '10), of Merrow, Conn., has been appointed Commissioner on Domestic Animals for Connecticut.

Dr. A. H. Hughes (K. C. V. C. '15) has resigned from his state position and gone into general practice at 1315 Third St., Corpus Christi, Texas.

Dr. A. E. Bott (Chi. '13), of East Saint Louis, Ill., is a member of the Board of Education of East Saint Louis and is now serving his second term.

Dr. E. E. Slatter (O. S. U. 30), who has been at the University of Illinois for the past three years, has located at Saint Joseph, Ill., for general practice.

Dr. J. G. Catlett (U. S. C. V. S. '16), of Miami, Fla., has been appointed a member of the Florida Board of Veterinary Examiners by Governor Sholtz.

Dr. F. R. Bartlow (Ind. '17), of Indianapolis, Ind., recently signed a long-time lease on the property at 839 N. Capitol Ave., for veterinary hospital purposes.

Dr. C. F. McKinney (T. H. '13), of Charleston, Ill., has purchased a piece of property on West State Street and will erect a modern veterinary hospital on it.

DR. G. W. SHIRLEY (Ga. '32) has taken over the practice of his brother, Dr. S. J. Shirley (Ga. '24), at Douglas, Ga., following the death of the latter.

DR. W. E. RUSSELL (Chi. '20), of Kitchener, Ontario, who specializes in the diseases of the silver fox, recently wrote that business was good in his territory.

DR. JOHN H. RUST (K. S. C. '32), who has been associated with Dr. F. F. Russell (Ont. '13-Corn. '16), at Corncord, N. H., the past year, is now located at Wellesley Hills, Mass.

DR. W. C. WOODRUFF (O. S. U. '16), of Cleveland, Ohio, was elected president of the Dog Owners Mutual Protective Association of America at a meeting held in Cleveland, on November 4.

DR. H. L. LYON (San Fran. '14), of Hillsville, Va., has been elected to the lower house of the Virginia legislature. His candidacy was commented upon in the September, 1933, issue of the JOURNAL.

DR. ALFRED L. BIRCH (Iowa '21), of Worthington, Minn., received a broken leg when the horse he was riding in a polo match at Paullina, Iowa, in October, collided with a horse ridden by another player.

DR. FRASER A. SMITH (Ont. '00), of Lexington, Ky., was called to California in October to examine Pillow Fight, noted New Zealand Thoroughbred belonging to David Davis, who owned the late Phar Lap.

DR. FREDERICK P. RUHL (Amer. '85), of Milford, Del., has sold his practice to Dr. Herbert G. Wohnsiedler (Corn. '33), after having conducted the practice continuously for 49 years. Dr. Ruhl's health would not permit him to round out 50 years in general practice.

DR. SIDNEY BOWMAN (Ind. '11), of Odon, Ind., was shot in the head accidentally by his 11-year-old son while quail hunting near Odon on November 18. Although about 50 bird shot entered his scalp, none pierced the skull.

DR. EDWIN CALLDEMEIER (Chi. '11), of Louisville, Ky., was severely injured in an automobile accident the latter part of October. He sustained a skull fracture as well as three broken ribs, but was recovering satisfactorily according to latest reports.

DR. H. J. ROLLINS (K. C. V. C. '16), of Rockingham, N. C., who has been a member of the Board of County Commissioners for more than five years, has been appointed Judge of the County Recorder's Court, to fill a vacancy caused by the death of Judge Barrett.

DR. J. D. HAWKINS (McK. '09), of East St. Louis, Ill., suffered severe injuries when his car overturned after a collision on the old Cahokia Road, on September 28. In an effort to avoid colliding with another machine, Dr. Hawkins swerved his car sharply, causing it to overturn.

DR. W. J. MURPHY (Amer. '96), of New York, N. Y., was elected a member of the State Legislature at the recent elections, defeating an opponent who had been in office for 19 years. Dr. Murphy is a practicing physician and at one time was in the service of the U. S. Bureau of Animal Industry.

DR. T. A. SHIPLEY (Chi. '90) retired from the service of the Bureau of Animal Industry, October 31, and, with Mrs. Shipley, he expects to spend the winter in South Texas. During September, Dr. Shipley was in charge of the AAA pig and sow slaughtering project of the Department of Agriculture, at Nebraska City, Neb.

DR. M. W. EMMEL (Iowa '19), who has been professor of animal pathology at the Alabama Polytechnic Institute for several years, has resigned to accept a position as assistant veterinarian at the Florida Agricultural Experiment Station, University of Florida, Gainesville. Dr. Emmel will devote his entire time to research work.